DOES NOT CIRCULATE

HUMAN BIOLOGY

a record of research

UNIVERSITY OF MICHIGAN
MAR 1956
MEDICAL LIBRARY

DECEMBER, 1955

Vol. 27 No. 4

PUBLISHED QUARTERLY BY
WAYNE UNIVERSITY PRESS, DETROIT 1, MICHIGAN, U.S.A.

HUMAN BIOLOGY

a record of research

FOUNDED BY RAYMOND PEARL

Editor

GABRIEL W. LASKER

in association with

BENTLEY GLASS — WILLIAM W. HOWELLS

JAMES N. SPUHLER — WILLIAM L. STRAUS, JR.

Review Editor

JOSEF BROŽEK

HUMAN BIOLOGY is published quarterly by Wayne University Press, Detroit 1, Michigan, U. S. A. The advance subscription price is $5.00 a year (foreign postage 35 cents; Canadian 15 cents). Single current issues are $2.00, plus postage.

Subscribers should notify the publishers of change in address at least two weeks before publication of the issue with which the change is to take effect. No claim for non-receipt of an issue will be entertained unless made immediately upon receipt of the next succeeding issue.

Manuscripts and communications regarding editorial matters should be addressed to Editor, Human Biology, Department of Anatomy, Wayne University College of Medicine, 1401 Rivard St., Detroit 7, Michigan.

WAYNE UNIVERSITY PRESS, *Publishers*

4841 Cass Avenue

DETROIT 1, MICHIGAN, U. S. A.

Entered as second class matter at the post office at Detroit, Michigan. Acceptance at the special rate of postage under section 34.40(e) P. L. & R. authorized.

Copyright 1955 by Wayne University Press

HUMAN BIOLOGY
a record of research

DECEMBER, 1955

VOL. 27 No. 4

BLOOD GROUPS, RACES AND PREHISTORY

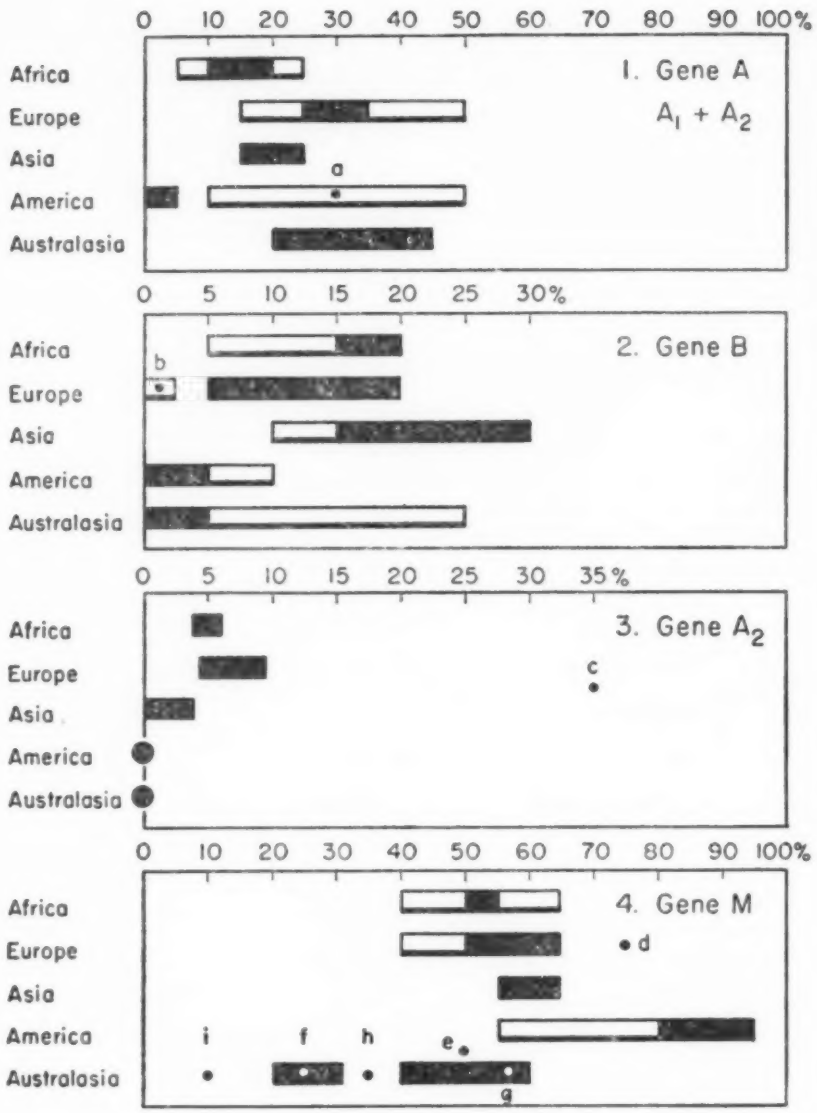
BY F. OTTENSOOSER

Laboratório Paulista de Biologia, São Paulo, Brazil

NOW that the vast material on blood group distribution over the world has been fully compiled by Mourant ('54) one may attempt to condense the outstanding data in a synoptic form. Short schemes as presented in this note necessarily disregard important details, but they should have didactic value and it will be seen that they lead, moreover, to interesting speculations.

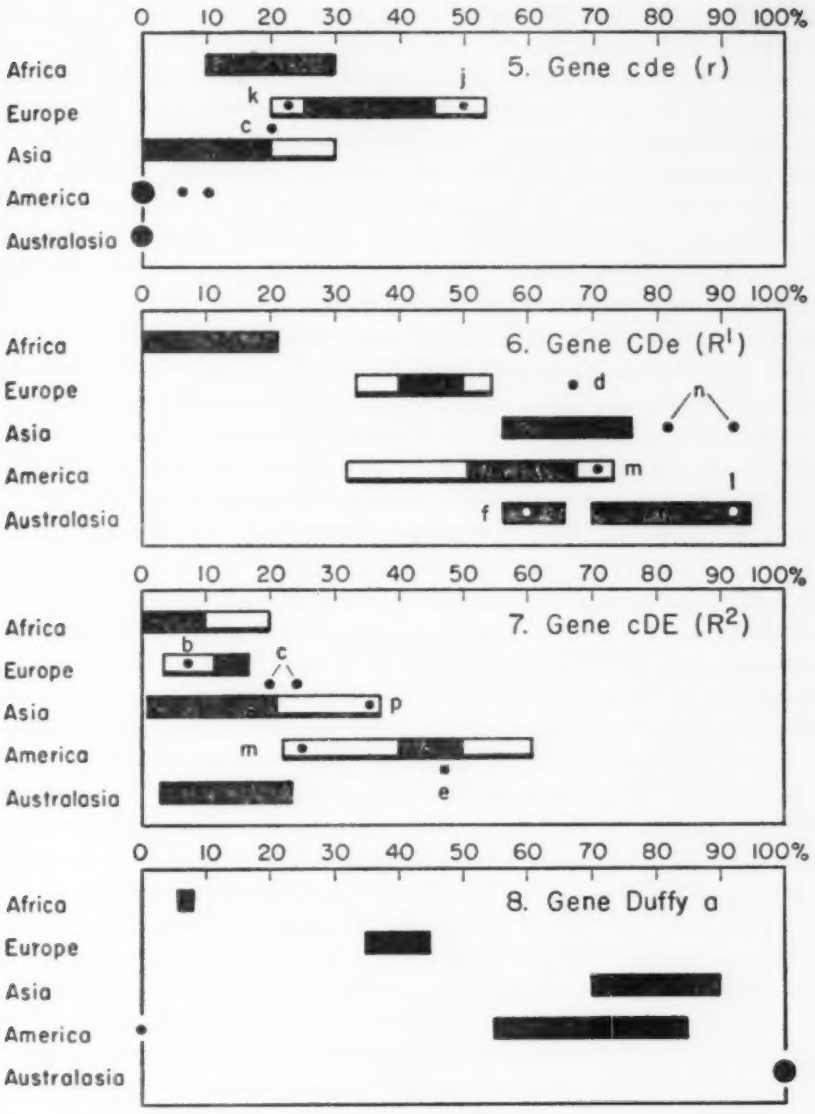
The schemes have in the main been confined to the 5 great races: the Negroes; the Caucasoids, including Mediterraneans as far as the Sahara and the Caspian Sea as well as Russians as far as the Urals; the remaining Asiatics, most of them typical Mongoloids; the American Indians and the Eskimos; the Australasians comprising the Australian aborigines and the populations of Oceania (Indonesians, Melanesians, Micronesians, Polynesians). Eight blood genes, comparatively well explored or yielding strong racial differences are considered.

There are numerous serological characteristics of the Negroes, three of which are shown in the schemes. They have less *CDe* (R^1) gene (below 25%) and less *Duffy* (Fy^a) gene (below 10%) than the other



FIGS. 1-4. BLACK PORTION OF BAR: MOST COMMON PERCENTAGES.

a. North American Indians. b. Basques. c. Lapps. d. Sardinians. e. Maori. f. Australian natives. g. Indonesians. h. Micronesians. i. Melanesians.



FIGS. 5-8. BLACK PORTION OF BAR: MOST COMMON PERCENTAGES.

b. Basques. **c.** Lapps. **d.** Sardinians. **e.** Maori. **f.** Australian natives. **j.** Basques, Berbers, Irish. **k.** Sardinians, Greeks. **l.** Australasians. **m.** Eskimos. **n.** Andaman Islanders. **p.** Japanese.

races; and the A_2 values of Negroes as of whites are surpassed only by those of the Lapps. Whites, specially the Basques, are rich in *cde* (*r*); however, this peculiarity is less marked in the Mediterranean area. Besides, no gene is known which would separate the whites from all other races at once, although they differ from each race in particular by several gene frequencies. Asia shows high frequencies of *B*, R^1 and *Duffy*. Some genes pass through Asia in gradients: The moderate values of *r* in the Mediterranean area seem to proceed with small variations as far as India, vanishing beyond this almost completely; A_2 decreases in the same direction, whereas R^1 goes the opposite way. The American Indians maintain the record of *cDE* (R^2) and together with the Eskimos also of *M*, but A_2 and, virtually, *r* are absent in both. The Australasians share with them the lack of A_2 and *r*, and have much R^1 and *Duffy*, the latter gene reaching sometimes 100%. There are sharp subdivisions, however. Indonesians still exhibit the high Asiatic *B* and *M* values. Melanesia and Micronesia are characterized by minimum of *M* and maximum of R^1. Australian aborigines, if pure, seem to lack *B* (and *S*). The Polynesians approach Amerindians in *M*, R^2 and *A*.

These well known data may exemplify the use of such schemes in teaching. Yet to take another point, they disclose some suggestive convergences, based on which we will try, with all reserve, to interpret some results of serological anthropology in a more general manner.

What at first attracts our attention is the similarity of the "profiles" of A_2 and *r* on the one hand and of R^1 and *Duffy* on the other. Among the 5 races the Australasians have the lowest values of the first two genes, and the highest ones of the other two. This configuration is hardly due to chance alone, since the probability of one extreme value of any gene for one out of five races is .4, and for the 4 genes, 0.4^4, about 0.026. In fact, the probability is even smaller because those 4 gene frequencies are in Australasians not merely higher or lower than in the other races, but approach or reach 0 or 100%.

Other variables than chance must be invoked to account for the accumulation of extreme values in Australasians. It clearly cannot be the result of mixing with any of the 4 other races. One could imagine that selection acted on *r*, selection or mutation on A_2, fluctuation on R^1 and *Duffy*. Be this as it may, mechanisms would have worked slowly always. Hence the association of 4 extreme values, together with other pecularities, points to an ancient race which originated in Australasia or Eastern Asia: the protomongoloid race.

This race would have been fixed before the separation of the American Indians, related to it by the frequencies of all those 4 genes. Possibly the Amerindians brought their high *M* and R^2 frequencies along from Siberia where data are lacking. Their high O frequency has less weight, *ABO* frequencies being less stable.

The Negroes form the opposite extreme to the protomongoloid picture regarding R^1 and *Duffy*, and they have in addition unique values for *ABO* secretion, *P* and particularly *cDe* (R^0). The probability of chance combination of five extreme values would be but 0.4^5, nearly 0.01. No scheme has been drawn for R^0 because part of the observations is not quite clear before extended by D^u tests. At any rate the high R^0 frequency in Negroes is so striking a feature that it alone suggests that the race is very old: the protonegroid race.

Serological evidence for the two old races may be correlated to some concepts of palaeo-anthropology. It has been claimed by several authorities that the ancient Javan forms (*Pithecanthropus* and Solo Man) can be linked with Australian natives of today, as well as the Rhodesian Man with living South African populations. On the other hand, no link has been found in Europe between Neanderthal Man and the modern Europeans. If anthropological characters of living people are traceable back to so remote an ancestry, it is not unlikely that blood gene frequencies may be more stable than is generally supposed. In any case the idea deserves consideration.

Gene frequencies of Europe and Asia, in contrast to those of Australasia and Africa, are mostly not extreme but intermediate, showing that repeated intensive mixture occurred even in historic times. Thus the white race appears comparatively young, as suggested also by the *B*-gradient, the high value of *r* and the very low value of $CDE(R^z)$.

Apart from Australasians and Negroes other smaller populations with exceptional blood group frequencies do exist. They have lived for long periods in isolation and they are in part "marginal" geographically. Accumulation of extreme values may be striking, as in Lapps and the Ainu, perhaps residues of ancient races.

In conclusion, it becomes probable that present blood group distributions can tell us something about prehistory.

LITERATURE CITED

MOURANT, A. E. 1954 The Distribution of the Human Blood Groups. Blackwell Scientific Publications, Oxford.

RETARDATION IN GROWTH OF CHILDREN IN EUROPE AND ASIA DURING WORLD WAR II

BY STEPHEN D. MARKOWITZ
Madison, Wisconsin

WORLD WAR II created an experimental situation for the study of the effects of wartime conditions on the growth of children in which environmental change provided an independent variable and consequences of this change, a dependent variable.

GROWTH OF CHILDREN

For the half century previous to World War II, there had been a tendency throughout the world for weight and height to increase within each age group from newborn to adult. This is clearly seen for age groups 13 to 18, for instance, in data compiled by the Japanese Ministry of Education (in Matsuyama, '48). This data is plotted in figures 1 and 2. The curves, except for a slight acceleration after 1921, rise steadily from 1901 to 1937, the last year used in this series. From this we can asume that the environment was changing at a fairly constant rate.

Curves made from data compiled by Matsuyama ('48) on a small group of boys are included on the same graphs for the years 1940 to 1946. The curves from 1901-1937 do not fit together with those of 1940-1946. Nevertheless, the two sets have been connected with dotted lines to indicate the corresponding age groups. Note the change of slope. In prewar years the curves are rising; in the war years the curves are falling. The curves for the war years, especially for height, are not steady, probably because of the small and inconstant number of children measured from year to year.

Similar curves were compiled by Solth and Abt ('51) for the weights of newborns from 1901 to 1948 in three German cities (Marburg, Berlin

and Wurzburg) and in one Swiss city (Basel). The curves for all 4 cities show the same change of slope at the start of World War II as the Japanese study except that the fall in the curve for the Swiss city is much less severe and takes place only during the first half of the war. The curves for the three German cities fall rapidly up to 1946, one year after the war. At this time they reverse direction and rise very rapidly toward their pre-war levels. In explaining this Solth and Abt discount all influences that are not connected with the environment.

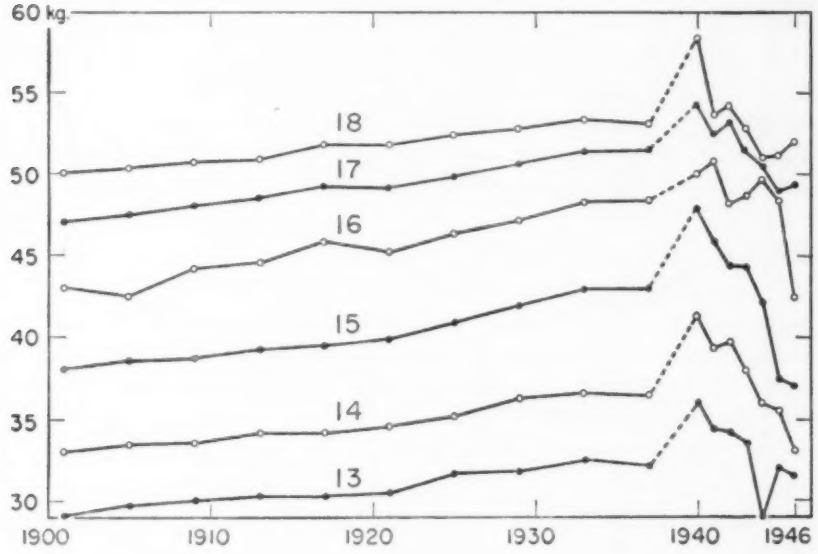


FIG. 1. WEIGHT OF JAPANESE SCHOOL BOYS FROM 1901 TO 1937; WEIGHT OF UENO MIDDLE SCHOOL BOYS, TOKYO, FROM 1940 TO 1946.

Another way of showing the effect of war environment on growth is to compare the heights and weights of children during the war with a pre-war standard. This method showed decreased height and weight in France (Douady and Trémolières, '47; Boulanger-Pilet, '46; Boulanger-Pilet *et al.*, '50; Laporte, '46; and Meiklejohn, '45), Germany (Stutte and Reinecke, '51), Greece (Valores, '46), Italy (Galeotti, '47), Belgium (Ellis, '45), China (Ni, '49), Finland (Kena-Wickstrand, '50) and Holland (Streng, cited by Hirsch, '46).

France was among the worst affected countries. In studies made in Paris, Marseilles, Nice, Lyon, Toulouse and Montpellier by several dif-

ferent investigators, the general outcome was a large reduction in weight and height during the war. The largest and most persistent reductions occurred among the age groups in which puberty usually begins (11-14 years of age). This retardation can be correlated with the delay of the onset of puberty that occurred generally during the war.

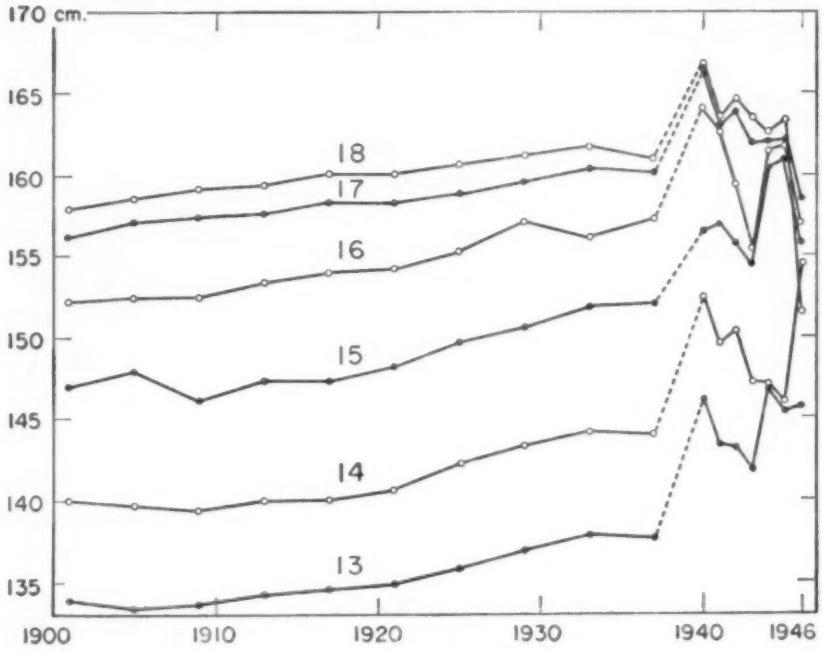


FIG. 2. HEIGHT OF JAPANESE SCHOOL BOYS FROM 1901 TO 1937; HEIGHT OF UENO MIDDLE SCHOOL BOYS, TOKYO, FROM 1940 TO 1946.

I have been informed by a French friend that food shortages in France were most acute in 1942. The food situation got progressively better in 1943, 1944 and 1945. One author, Meiklejohn ('45), states that the food situation in 1945 was up to the standards of Great Britain, while Douady and Trémolières ('47) state that nutrition, although improved, was still insufficient in 1945.

Boulanger-Pilet, Delagrange, and Trémolières ('50) compared measurements taken on the children of the *Écoles Primaires de la Seine* in the years 1938-39, 1942, 1946, and 1948. The measurements were compiled for girls and boys aged 6 to 15. The percentage changes in

height and weight for the years 1942, 1946, and 1948 using 1938-39 as a standard year, are included in tables 1 and 2. The reductions in height in 1942 are very slight and take place for boys between the ages 11 and 13 (amout 1.2% reduction) and for girls between the ages of 8 and 11 (about 0.8% reduction). During the same period the boys in the 10- and the 14-year age groups had increased in height (0.8% and 0.7% respectively); the girls in the 6-year group had increased 1.8% in height. The reductions in weight were greater and not as questionable as were the reductions in height. There were reductions in weight averaging 2.9% for boys, excluding the oldest group and 3.0% for girls, including all groups. The largest reductions occurred in the older groups (in boys, age 13, 5%; in girls, age 11, 4.5%—see table 2, column 1) as in all studies where reductions in weight have taken place. There are increases in 1946 and 1948; the older groups are the slowest to recover from the deficits and girls seem to improve faster (tables 1 and 2, columns 1, 2 and 3).

I have summarized this last study in detail since it shows the least retardation in growth of any study published on French children. The reductions in height are questionable since they are small or non-existent. In respect to height, at least, this study compares with one from England (Bransby, '46) and one from Italy (Metcoff and McQueeney, '46) which did not find reductions in height and weight. Does this mean that the war environment affected only the weight of the Seine school children and not the height, and that in England and Italy there was no change in growth?

It is not necessary to find absolute reductions in height and weight to conclude that the war environment affected the growth of children. The prewar Japanese curves, or better, those for Holland (Streng, cited in Hirsch, '49) demonstrate a change in the secular trend. The average height of Dutch children tended to increase from 1938 to 1941. This increase of 6 cm was partly lost (1 to 2 cm) during the war years. Here one finds absolute reductions during the later war years if 1941 is taken as a standard of comparison but absolute increases if 1938 is taken. Thus, apparent increases in height or weight do not necessarily mean that the growth patterns were not retarded. To show that there was no change one would have to draw a continuous height and weight curve for the prewar and postwar years which did not change its slope. Furthermore, failure to change in weight probably also indicates a change in the conditions of growth if there had previously been steady year to year

TABLE 1.

Height of Children during World War II compared with Pre-war Standards (percentage differences)

COUNTRY	FRANCE												GERMANY		GREECE				ITALY		BELGIUM			CHINA		JAPAN
AUTHOR	BOULANGER-PILET, *et al.*						DOUADY AND TRÉMOLIERÈS				LAPORTE		STUTTE & REINECKE HESSE		VALORAS				GALEOTTI		ELLIS			NI		MATSUYAMA
PLACE	SEINE DISTRICT						MONTPELLIER				PARIS		PRE WAR		ATHENS				ROME		BRUSSELS			SHANGHAI		TOKYO
STANDARD	1938-39						1938				1938				1927-28				1939-41		1938-39		1938	1939		1940
COMPARISON	1942		1946		1948		1942	1944		1945	1944		1946		1942-43		1945		1945		1944		1944	1945		1945
	1		2		3		4	5		6	7		8		9*		10*		11		12		13	14*		15
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♂	♀	♂
AGE																										
4																								−2.8	−5.8	
5																								−4.0	−3.8	
6	0	+1.8	0	0	+0.9	+0.9			−2.0	−0.2	−1.2	−0.4	−5.4	−1.6	+1.7	+4.1					−1.3	−0.5			−5.0	−6.0	
7	0	0	+0.8	+0.9	+1.7	+1.7			−1.7	−1.1	−1.5	−0.6	−1.5	−3.3	+1.8	+2.9					−1.4	−0.8			−5.1	−5.5	
8	0	−0.8	+0.8	0	+1.6	+0.8	−2.3	−4.1	−1.6	−0.5	−1.0	+0.9	−3.6	−2.9	+0.9	+2.4	+3.8	+3.6	−1.8	−0.3	−3.1	−1.3			−2.9	−3.4	
9	0	−0.8	0	0	+1.6	+0.8	−3.1	−2.3	−3.0	−0.9	−1.6	+0.4	−0.4	−2.4	+0.9	+1.9	+1.5	+3.0	−1.6	−0.2					−2.2	−2.7	
10	+0.8	−0.8	0	−0.8	+1.5	+1.5	−1.5	−1.5	−1.1	−0.2	+0.2	+0.8	−0.6	−2.4	+1.0	+1.6	+2.4	+1.4	−2.1	−0.2	−0.8	−1.2			−1.9	−3.7	
11	−1.5	−0.7	0	0	+0.7	+1.4	−3.6	−3.3	−3.4	−2.2	−3.1	−1.8	−5.5	−1.1	+0.6	+0.6	+2.0	+1.9	−0.3	−0.7					−4.5	−5.8	
12	−1.4	0	−0.7	−0.7	+0.7	+1.4	−3.5	−4.9	−3.1	−0.5	−2.9	−3.4	−2.6	−2.6	−0.1	−0.3	+3.6	+2.0			−2.7	−4.2					
13	−0.7	0	−0.7	0	+1.4	+1.4	−2.7	−2.7	−1.7	−2.7	−2.3	−3.4	−3.5	−0.9	−0.5	−1.1	+2.4	+2.7	−1.8	−1.7							
14	+0.7	0	+1.3	+1.3	+2.7	+2.7	−6.0	−0.8		−1.0	−5.8	−1.5	−2.6	−1.8	−1.1	−0.1	+1.8	+2.6	−3.6	−2.7	−2.2	−0.7					−0.4
15													−1.3	−1.6			+1.3	+1.0					−2.6				−4.2
16													−0.9	+1.3			+0.7	+1.9					−1.0				−2.9
17																	+1.1	+2.9					−0.2				−1.4
18																	+2.0	+1.9					−0.8				−2.6
																							+0.2				**−2.1**

* Age to nearest birthday.

TABLE 2.

Weight of Children during World War II compared with Pre-war Standards (percentage differences)

COUNTRY	FRANCE												GERMANY		GREECE				ITALY		BELGIUM			CHINA		JAPAN
AUTHOR	BOULANGER-PILET, *et al.*						DOUADY AND TRÉMOLIÈRES						LAPORTE		STUTTE & REINECKE HESSE		VALORAS		GALEOTTI		ELLIS			NI		MATSUYAMA
PLACE	SEINE DISTRICT						MONTPELLIER						PARIS		PRE WAR		ATHENS		ROME		BRUSSELS			SHANGHAI		TOKYO
STANDARD	1938–39						1938						1938				1927–28									
COMPARISON	1942		1946		1948		1942		1944		1945		1944		1946		1942–43	1945	1939–41	1945	1938–39	1944	1938	1939	1945	1940	1945
	1		2		3		4		5		6		7		8		9*	10*	11		12		13	14*		15	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♂	♀	♂
AGE																										
4																										
5																								−2.0	−3.9	
6	−1.4	−1.8	+0.7	+1.0	+1.3	+1.0			−7.2	−7.4	−2.9		−15.2	−4.4	+2.9	+5.1								−3.9	−7.0	
7	−2.1	−2.8	+1.0	+2.6	+1.7	+2.2			−9.7	−5.0	−4.8	+5.0	−1.2	−3.0	−0.9	+3.2			−0.5	−2.9				−5.1	−8.5	
8	−2.4	−2.3	+1.4	+1.4	+0.9	+2.5	−4.0	−16.5	−2.0	−8.3	+0.4	+1.2	−7.3	−6.0	−1.2	−0.4			−0.5	−4.4	−8.3	−8.8		−3.4	−7.6	
9	−1.1	−3.7	+0.1	+0.4	+1.3	+1.2	−7.3	−7.4	−7.3	−5.9	−1.1	0	−5.1	−4.8	−1.4	−1.1	−12.2	−13.3	−3.5	−2.0				−4.6	−5.7	
10	−2.6	−4.5	+0.4	−0.8	+1.6	+1.7	−10.0	−6.7	−10.1	−5.4	−3.4	+0.3	−5.0	−10.2	−2.0	−4.5	−9.0	−9.4	−3.5	−1.9	−1.8	−4.5		−4.5	−3.7	
11	−4.3	−4.7	−0.1	+0.7	+0.4	+1.5	0	−13.6	−7.7	−7.6	−3.1	−5.7	−4.8	−4.0	−2.4	−5.0	−3.0	−12.5	−4.6	−1.3				−2.6	−3.0	
12	−4.5	−3.0	−1.8	−1.1	−0.3	+2.6	−16.6	−16.3	−15.2	−10.0	−12.5	−8.7	−9.5	−8.7	−4.5	−8.0	−0.6	−14.9	−3.1	−2.8	−6.1	−13.6		−7.6	−14.3	
13	−5.0	−3.7	−4.0	+1.2	−0.3	+3.7	−14.8	−12.2	−11.1	−10.2	−10.1	−7.3	−4.8	−3.3	−5.2	−7.8	−4.2	−9.8								
14	+0.8	−0.7	+4.6	+6.1	+7.5	+7.9	−12.0	−7.0	−16.8	−8.4	−10.2	−5.3	−11.2	−2.2	−7.8	−7.9	−9.5	−8.9			−5.4	−1.6				−11.1
15													−6.8	−2.3			−11.6	−9.6					−10.7			−13.8
16													−7.1	+3.6			−5.4	−2.3					−7.9			−21.8
17																	−0.3	+2.8					−4.8			−3.2
18																	+0.2	+3.2					0			−9.8
19																		+0.3					−0.2			−12.2
																	+5.4									

*Age to nearest birthday.

increases. Decreases in height and weight can be taken as more positive evidence for changes in growth since the heights and weights of pre-war times certainly were not decreasing. Finally, one can easily see that the best years to use as standards of comparison are those closest to the war years. Decreases in height in France might have appeared greater if our standard of comparison had been taken around 1940 instead of 1938-39, since the former year might have had maximum values.

Two reports, one by Laporte ('46) and the other by Boulanger-Pilet ('46) deal with reductions in height and weight of Paris school children in 1944. Both seem to deal with the same group of children, but Laporte deals with ages 6 to 16 and Boulanger-Pilet, with ages 6 to 17. The standard of comparison is 1938. The percentage differences calculated from data presented in Laporte's article is included in tables 1 and 2, column 7. The maximum percentage differences in height occur for boys at the age of 11 (5.5%) and for girls at the age of 7 (3.3%) (table 1). The maximum percentage differences in weight occur for the boys at the fourteenth year and for girls at the tenth year (10.2%) (table 2); both sexes have large differences in the twelfth year. Boulanger-Pilet notes also a large difference in weight of boys at the seventeenth year (4 kg, about 8%). Laporte ('46, p. 247) explains the increase in the weight and height of 16-year-old girls by stating that "most of the older girls worked on farms during their vacations and thus obtained the benefit of better food." Boulanger-Pilet gave a similar explanation.

Douady and Trémolières ('47) in a study of children aged 8 to 14 from Paris, Marseilles, Strasbourg, Toulouse and Montpellier in 1945 found an average deficiency of 7 cm (about 4.7%) and 5 kg (about 11.6%) for the girls of 14 years, and 4 cm (about 2.7%) and 6 kg (about 14.4%) for the boys of the same age. In the 13th *arrondissement* of Paris, where the standard of life is low and where there were large deficiencies in weight and height in 1944, the reductions of 1944 had been halved in both height and weight for all age groups by 1945. In the 18th *arrondissement* of Paris, where the reductions in 1944 were nearly half those of the 13th *arrondissement*, the reductions in height are halved in 1945 but the reductions in weight remain unchanged. Douady and Trémolières relate these improvements and the improvements to be seen in Montpellier (tables 1 and 2, columns 4, 5 and 6) to the improvement in nutrition in 1945. Douady and Trémolières did not present any tables, only graphs. Since the graph for the city of Mont-

pellier showed the differences in growth over three years, however, I felt it worthwhile to try to interpret the percentage changes from them. The graphs showed the differences in 1942, 1944, and 1945, taking 1938 as a base line. In estimating the changes I used the figures for Seine school children for the year 1938-39 as a base.

Meiklejohn ('45) in studies in Marseilles, Lyon, Nice, and Paris concludes that French children over 7 years of age show evidence of growth retardation. Stuart (cited by Ni, '47, p. 377) "concludes that probably half of the [French] children of school age show a retardation in growth."

Stutte and Reinecke give the height and weights of Hessian school children for the years 1946 to 1949. They include the data of Pfaundler which were taken as the pre-war German standards. Actually, Pfaundler's data, taken from the general German population, cannot properly be compared with data limited to Hesse. But the percentages for 1946 have been included in tables 1 and 2 to indicate the differences. The heights of the younger Hessian children increased in 1946 over those of pre-war German youth. In interpreting this, one should recall: (1) The conclusion of the French studies where the positive increments did not disprove a change in growth pattern; (2) younger children are the quickest to recover from the growth deficiencies; (3) the Hessian measurements were made one year after the war when environmental improvements would have occurred; and (4) there is no year given for Pfaundler's data. The Hessian weights are generally reduced except for the very youngest age groups (6 and 7). Note the trend in the weights as one goes from the youngest to the oldest age groups. The deficiencies increase as one approaches the years at which puberty usually occurs. This trend is also seen in the heights where the increases reach a maximum, decrease, and finally become negative.

Stutte and Reinecke also give a summary of data for the rest of Germany. They cite Mayer who found that the growth of 12-15 year old Hamburg children was severely hampered during the war. On the whole, Hamburg children were more severely retarded in growth than Hessian children, the retardation starting at the 6-year age group. They also mention that in 1946 Roninger found in Schleswig-Holstein a deficit of 5–7 cm in urban and rural children and that, in Solingen in 1945, Adam found that school children were 1–2 kg and 2–3 cm below the averages of pre-war children.

Some of the data for Greece (Valoras, '46), have been put in tables

1 and 2, columns 9 and 10, as percentages. These data do not give an accurate picture of the change in height growth during the war because standard values based on measurements made in 1927-28 may differ considerably from what would have been found immediately before the war. If one used the standard for the Seine school children, one would get all negative percentages for the age groups between 8 and 13. The weight values give a truer picture of the change in growth that followed "the country-wide famine of 1941-42. . . Measurements from the 1945 survey indicate that the continued undernutrition had affected height growth as well as weight and the children were shorter and lighter than those of similar age measured two to two and one-half years previously" (Valoras, '46, pp. 215 and 228). Thus by 1945 height was definitely affected and Valoras points out that it was probably affected in 1942-43 by considering "the consistent decline in height increase for boys from age 11 to 17 years, normally a period of rapid growth" (Valoras, '46, p. 228).

One study in Italy failed to find changes in growth (Metcoff and McQueeney, '46). We have already concluded, however, that a constant pattern probably represents a change from a pattern which is graphed as a rising slope. For another Italian study which reveals growth reductions see tables 1 and 2, column 11. In addition to the reductions in weight and height, thoracic circumference was reduced 3.2 and 4.4% in males and females respectively between the ages of 6 and 11. Galeotti ('47, p. 295) concludes: "Such deficiencies should be for the average of children in Rome really greater than appears from the above figures inasmuch as the children studied in 1939-41 belonged to the poorest social classes. The growing conditions of such children are in fact normally worse than those relating to a great number of children chosen at random (as it is in the 1945 survey)." This would imply that the wealthier social classes had better environmental conditions for growth during as well as before the war. In the case of nutrition, this was true in certain other countries as well. A Hong Kong girl relates that the rich were always able to get food during the worst shortages of the Japanese occupation. Ellis ('45) and Meiklejohn ('45) tell about the black markets that operated in Belgium and France, respectively, during the war and for a period after. However, Boulanger-Pilet ('46) notes that some of the poorest Parisian families lived on the outskirts of the city and had the advantage of bicycling to the nearby country for food.

Ellis ('45) found reductions in height and weight of the children

of the working-class community of Andelecht in Brussels. The comparison year is 1944 and the standard is a random sampling of the community for 1938-39. Since the percentages are based on data that is subject to sampling errors they only "can be taken as indicating . . . an average diminuation of both height and weight in 1944 as compared with 1938-39 . . ." (Ellis, '45, p. 103). Ellis also compared the heights and weights in Andelecht for the years 1942, 1943, and 1944. He found that the "working-class children living at home have shown a gradual improvement in weight from 1942 to 1944, and that their height (with the possible exception of those reaching the normal age of puberty during the war) has been little affected." 1942 was the year of serious food shortages. By 1944, these shortages were corrected to some extent "by distribution of herrings, by an increased supply of potatoes, and finally by organization of the black market." (Quoted by Ellis from Bastenie and others).

Ellis follows the growth of 10 girls from the age of $7\frac{1}{2}$ in 1939 to $12\frac{1}{2}$ in 1944. The height and weight curves are graphed along with the standard of 1934 (compiled in the "period of severe economic depression in 1928" but published in 1934) for the corresponding ages. The height and weight curves follow the 1934 standard until 1942 (girls at the age of $10\frac{1}{2}$) and then fall below the standard in 1943 and 1944, the last year computed, when the girls were aged $12\frac{1}{2}$. Ellis indicates severe deficiencies in height and weight of employed telegraph boys (column 13) in 1944 as compared to the 1938-39 standard for employed boys. These deficiencies take place between 14 (the earliest year computed) and 16 and they amount to 2.6% in height and 10.7% in weight for the 14-year olds. Finally, he concludes that "adolescents . . . suffered more than the younger age group and a study of 106 adolescent girls indicates a retardation of puberty in a significant proportion of those living at home. . . ." Further, he shows by the device of three superimposed weight curves that there was a "marked delay in 1942 (following the severest period of rationing), a moderate delay in 1943 and an approximately normal onset of puberty in 1944."

Ni ('49, p. 680) found reductions in the heights and weights of "over 1,000 Shanghai school children 4 to 11 years of age, in 1945 . . . [as compared with] those of school children studied in 1939 for the corresponding age groups . . ." (tables 1 and 2, column 14). In this case it seems that the children aged 5, 6, and 7 and those aged 11 were the worst affected. Ni ('47, p. 378) notes that the 6- and 7-year olds are

"more interested in games and muscular activities and, therefore, require more food than in younger years [while at the same time] . . . their actual intake of food is more dictated by the taste, flavour and appearance of a meal, by the emotional state of the children, and by the patience, experience, and tact of the parents than either by the body requirements or by the parents' instruction. . . ." This may be the reason why the younger groups show retardations of the same magnitude as the oldest group. In European countries, although the rationing practices favored the younger groups, large deficiencies can sometimes be found in the 6- and 7-year olds. For instance, see column 7 of tables 1 and 2.

In column 15 of tables 1 and 2 the percent differences for Japanese boys (Matsuyama, '48) are represented. These differences are based upon the data from which figures 1 and 2 were made except that only the years 1940 (the standard year) and 1945 (the comparison year) are used. This column forms a basis of comparison between the continuous curve and percent of absolute difference methods. Matsuyama also gives data for thoracic circumference for the entire 13- to 18-year old male Japanese population from 1901 to 1937 and for a small group of 13- to 18-year old Tokyo boys from 1940 to 1946. The percentage decrease in 1945 as compared to the standard year 1940 averaged 3.2% with a maximum decrease in the 13-year olds (8.3%).

BIRTH WEIGHT

In table 3 we have the percentage reductions in birth weights for Holland, Russia, Germany, and a neutral country, Switzerland. The percentages listed under Solth and Abt were calculated from estimates derived from the curves which they published.

Leningrad, Russia, seems to have been one of the most severely affected, having maximum differences of 18.3% and 16.4% for males and females, respectively, in the period January-June 1942, one year from the standard. These decreases were noted during the siege of Leningrad (1941-1942) by Antonov ('47, p. 254):

> **The fall in the average birth weight was much greater in the first half of 1942, that is among mothers who suffered from hunger during all or nearly all of their periods of pregnancy. . . . Almost equal decrease in average birth weight of children carried to term was observed in other maternity clinics in Leningrad. In the Sneginev clinic, the average decrease was 500 gm (about 15%). In the obstetric-gynecologic clinic of the Second Leningrad Medical Institute the average weight in 1942 was 410 gm less than in 1940.**

Dean ('50) found in Wuppertal, Germany a reduction 185 gm (about 5.4%); the shortening of the gestation period could account for 45 gm (about 1.3%). This reduction in birth weight took place "in 1945 when the food situation was at its worst."

Smith ('47, p. 236) finds 8-9% reduction in the weight of Rotterdam newborn in 1945 compared to "the weight in normal times, the percentage being only slightly greater in the heavier infants . . . than in the light ones. . . ." Normal times presumably means the pre-war winter 1938-39 in the same hospital in Rotterdam. In table 3 I use a war period immediately preceding the period of "nutritional stringency," and therefore the percent difference shown is a smaller value (7.1%). Schaible ('49) noted a 7.3% reduction in newborn of Reutlingen, Germany for 1945-1947 as compared to 1938-1942. Belgium and Italy seem to be exceptions as far as absolute differences in birth weight are concerned (Ellis, '45; Pozzan, '51).

The general conclusion of all the investigators was that retardation in growth occurred during the war, but none of the investigators were successful in defining the specific factors which controlled the change in growth. The most they did was to describe the events of war and state that these events affected the development of the child. In attempting to classify environment, one is only concerned with that part of the environment that affects growth. We must link the specific parts of the environment with the body reactions.

To do this, one could start with the body reactions themselves. All body reactions can be equated with the total energy of the organism. This total energy encompasses the energy of growth and the energy needed to keep alive. The energy of growth is of secondary importance to the energy needed to keep alive since an organism must be alive in order to grow. Therefore, in times when the energy produced by the body is lower than normal, the energy of growth would be expected to suffer for the sake of conservation of adequate energy to keep alive.

If the war data is reviewed, it will be seen that events were correlated with reductions in weight and height. One of these, the increase in the age at which puberty begins, has been noticed by several investigators (Ellis, '45, Douady and Trémolières, '47). The energy needed to cause the physiological changes that occur with the onset of puberty is energy of growth. During the war the lack of food and the inefficient use of existing food, due to psychological conditions, would produce a limited amount of body energy. What energy was produced by the body would

TABLE 3.

Birth Weight of Infants Born during World War II compared with Pre-war Standards (percentage differences)

COUNTRY AUTHOR CITY STANDARD COMPARISON	RUSSIA ANTONOV LENINGRAD JANUARY—JUNE, 1941 JUL.—DEC. '41 JAN.—JUN. '42 JUL.—DEC. '42						HOLLAND SMITH ROTTERDAM OCT. 1—NOV. 15, '44 NOV. 16, '44— FEB. 14, '45	DEAN WUPPERTAL 1937–38 1945	GERMANY SOLTH AND ABT			SCHAIBLE REUTLINGEN 1938–42 1945–47	WITZERLAND SOLTH & ABT BASEL 1939 1945
									BERLIN 1939 1945	MARBURG 1939 1945	WURZBURG 1939 1945		
	♂	♀	♂	♀	♂	♀							
	−3.0	−2.4	−18.3	−16.4	−7.1	−12.5	−7.1	−5.4	−2.1	−3.2	−2.2	[−7.3	−0.8

be used in keeping the body alive, not in bringing about the onset of puberty. In many countries, notably Greece, Holland, and Russia, 50-70% amenorrhea was prevalent during the worst food shortages (Antonov, '47; Smith, '47; Valoras, '46). As in the case of pubescent growth, the decline in menstruation cycles would conserve body energy in the female.

If we begin to understand the processes of reactions within the body, we can proceed to look for the specific stimuli of these energy producing reactions. As in the case of the body reactions, we can divide the environmental stimuli into primary and secondary. Under primary we would include such things as nutrition and respiration since these are necessary for life. Hirsch ('49) in his study of Jerusalem children during the Arab-Israeli War also mentions the amount of sunlight as a factor. Psychological phenomena would be secondary.

We now begin to see the inadequacy of the experimental situation created by World War II. We have correlated the reduction in growth with a reduction in body energy output. However, it is impossible with the present data to separate the effects of nutritional and psychological stimuli on wartime growth. The relative importance of the two stimuli is unknown. Most of the workers considered the nutritional stimulus. Nutrition may be the most important factor, but to consider nutrition alone is to disregard the individual as a dynamic organism responding to stimuli other than fuel. Psychological factors may also have been involved. Conditions of war create stress and this may affect appetite, efficiency of digestion and absorption, and efficiency in utilization of absorbed food. Banning (cited by Keys *et al.*, '50, p. 92) ascribed the weight loss in the population of Holland in the first months of World War II to psychological factors "since the food supply was not greatly deficient."

The heights and weights of English school children generally remained constant during the first years of World War II. If the English heights and weights were increasing in pre-war times, this constancy would indicate a change in growth patterns. And, if this be true, this change cannot be correlated with nutrition since England suffered no more than a "slight underfeeding" (Keys *et al.*, '50, p. 17). From 1943-1945, heights and weights of English school children were above pre-war levels (Bransby, '46). This may indicate an adjustment to the psychological stress.

SUMMARY

World War II has provided us with an experimental situation for the study of growth. Although few studies permit one to draw a continuous curve of growth throughout this period for a homogeneous group of children over a span of time, several permit one to plot two or three points and to consider absolute differences. From studies of heights and weights of children and weights of newborn infants in Japan, China, England, France, Germany, Switzerland, Italy, Greece, Belgium, Holland, Finland and Russia one reaches the general conclusion of a retardation in growth during World War II. The role of nutritional (part of the primary stimulus) and psychological (part of the secondary stimulus) factors cannot be separated, however. Although it is clear that nutrition affects growth, it is not possible to isolate the factor of food shortage in the war situation. When and where food was scarcest, growth was usually most retarded, but there, also, conditions for emotional upset were at a maximum. The importance of psychological factors is still to be determined.

ACKNOWLEDGMENTS

I am indebted to Dr. Gabriel Lasker for suggesting the topic of this paper and for the use of the bibliographies which he has published in the *Yearbook of Physical Anthropology* during the post-war years. I am also indebted to Miss Helen Crawford, librarian at the University of Wisconsin Medical School, for aid in obtaining copies or microfilms of most of the literature cited.

LITERATURE CITED

ANTONOV, A. N. 1947 Children born during the siege of Leningrad in 1942. J. Pediat., *3*: 250-259.

BOULANGER-PILET 1946 Poids et tailles des écoliers parisiens en 1938 et en 1944. Arch. Franç. Pédiat., *3*: 101-102.

BOULANGER-PILET, DELAGRANGE AND TRÉMOLIÈRES 1950 Evolution de la croissance des enfants des écoles primaires de la Seine de 1939 à 1948; les chiffres actuels de taille et de poid. Ibid. 7: 711-719.

BRANSBY, E. R. 1946 Further note on the wartime growth of schoolchildren. Monthly Bull. Ministry of Health and Emergency Publ. Health Lab. Service, *5*: 57-60.

DEAN, R. F. A. 1950 The effect of undernutrition on the size of the baby at birth and on the ability of the mother to lactate. Proc. R. Soc. Med., *43*: 273-274.

DOUADY, D., AND J. TRÉMOLIÈRES 1947 Persistances d'importants déficits de croissance chez les infants d'âge scolaire en 1945-1946. La Press Médicale, *55*: 599-600.

ELLIS, R. W. B. 1945 Growth and health of Belgian children during and after the German Occupation (1940-1944). Arch. Dis. Child., *20*: 97-109.

GALEOTTI, G. 1947 Lo sviluppo somatico degli alumni delle Scuole Communali di Roma in periodo di emergenza alimentare (1945). Rivista di Antropologia, *35*: 285-295.

HIRSCH, W. 1949 Growth in infants under abnormal conditions (Observations during siege of Jerusalem in summer 1948). Annales Paediatrici, *172*: 339-343.

KENA-WICKSTRAND, LEMPI 1947 Did the recent wars affect the children's growth in height? Annales Med. Internae Fenniae, *36*: 526-530.

KEYS, A., *et al.* 1950 Biology of Human Starvation. University of Minnesota Press, Minneapolis, 2 vols.

KUO, J. T., AND T. G. NI 1950 Analysis of physical measurements of over 15,000 Shanghai students. Chinese Med. J., *68*: 164-168.

LAPORTE, M. 1946 Effect of war-imposed dietary limitations on growth of Paris school children. Amer. J. Dis. Child., *71*: 244-247.

MATSUYAMA, H. 1948 The physique of Japanese middle school boys during the war years. Research Quart. Amer. Assoc. for Health and Physical Education, *19*: 30-33.

METCOFF, J. AND A. J. MCQUEENEY 1946 Clinical malnutrition in Italy in 1945. New Engl. J. Med., *235*: 451-460.

MEIKLEJOHN, A. P. 1945 Report on the state of nutrition in France 1941 to January 1945. Mimeographed. Cited from an abstract by H. E. Harding, Bull. Hyg., *20*: 624-625.

NI, T. G. 1947 Heights and weights of Shanghai school children. Chinese Med. J., *65*: 373-380.

——— 1949 The physical growth of Shanghai school children. Chinese Med. J., *67*: 677-680.

POZZAN, M. 1951 Rilievi statistici sull'accrescimento ponderale del lattante padovano nell decennio 1938-1947. Minerva Pediatrica, *3*: 134-135.

SCHAIBLE, G. 1949 Ernährung und Geburtsgewicht. Deutsche Med. Wochenschrift, *74*: 144-146.

SOLTH, K., AND K. ABT 1951 Die Veränderungen des Geburtsgewichtes in den letzten fünfzig Jahren. Vergleich deutscher Kliniken mit dem Frauenspital Basel. Schweizerische Medizinische Wochenschrift, *81*: 58-61.

SMITH, C. A. 1947 Effects of maternal undernutrition upon the newborn infant in Holland (1944-1945). J. of Pediat., *30*: 229-243.

STUTTE, H. AND H. REINECKE 1951 Wachstumsverhältnisse bei hessischen Schulkindern in den Jahren 1946 bis 1949. Kinderärztl. Praxis, *19*: 515-521.

VALORAS, V. G. 1946 Effects of famine on the population of Greece. Millbank Memorial Fund Quart., *24*: 215-234.

FINGERNAIL GROWTH IN THE HUMAN SUBJECT

RATES AND VARIATIONS IN 300 INDIVIDUALS

BY ROBERT W. HILLMAN

Department of Environmental Medicine and Community Health
State University College of Medicine at New York City

GROWTH, other than neoplastic, is probably one of the more acceptable attributes of health. But, except for the physiological phenomena of pregnancy and the reparative responses to stress, growth—as distinct from development—is not a measurable characteristic of most "normal" adult human tissues. The maintenance and replacement of epithelial surfaces, however, is at least a related activity, and the skin provides an accessible area for observing this process. Its variable appendages, the hair and nails, have been intensively studied by anthropologist and clinician alike, as evidenced by numerous published descriptions of their characteristics in health and disease.

These observations have been almost exclusively qualitative—particularly as concerns the fingernails. Moreover, most of the occasional, recorded quantitative nail growth studies have related to only a few or even single patients with local or systematic disorders. Sharpey-Schafer ('30), Wigand ('37), Sunderland and Ray ('52), and, in this country, Edwards ('38) are among the principal recent contributors to this limited available information. Beans' ('53) 10 year self-study constitutes a uniquely long series of observations, while the largest collections of data on the subject appear to have been assembled by Clark and Buxton ('38) and Gilchrist and Buxton ('39). Although these last involved a relatively narrow age range (10 to 23) over evidently short periods of time, they seem the most authoritative basis for current concepts of nail growth rates in the normal human subject.

The present inquiry was designed to provide further information concerning the growth rate of fingernails, particulary to document intra- and inter-individual variations and to determine the possible influence

of such factors as age, sex, intercurrent illness and environmental change. A second objective was related to the concept that significant departures from established growth patterns might reflect pathological states and that the practical demonstration of these irregularities might facilitate the early detection or the rate of progression of disease.

MATERIALS AND METHODS

Fingernail growth was measured in 300 individuals, including 148 medical students and 63 student nurses. The remaining subjects were predominantly professional, technical and secretarial personnel, supplemented by two family groups. In all, 293 subjects were considered ostensibly "normal." Of the others, two had diabetes mellitus, two rheumatoid arthritis (one on intermittent steroid therapy), two peptic ulcer syndrome (one post-gastrectomy), and one an active, chronic suppurative osteomyelitis. Relevant personal data were provided by questionnaires, supplemented in most instance by health records.

There were in the study 179 males and 121 females, including one set each of identical and fraternal twins. The age range was 4 to 67 with a median of 21 and a mean of 25.6 years. The average period of observation was 154 days, the median being 176 and the range 25 to 457 days. Ten individuals were observed for a period of at least one year. There was a total of 991 separate observation periods, ranging from two to 8 weeks, with an average interval of 46 days, providing a cumulative total of approximately 46,200 person-observation days. Since from two to 10 nails were measured for each subject, the number of nail-observation days was obviously greatly in excess of this figure.

Growth rate was determined by measuring the distal progress of a transverse mark, inscribed with an "ampoule" file at the convex margin of the lunula. A fine-pointed, "E.C.G." type calipers was employed to span the intervening space. The distance was then measured off on a blocked steel rule, and the result converted into a metric reading. All measurements were made by one of two observers, with readings being satisfactorily reproducible.

RESULTS

Results are summarized in tables 1 and 2 and in figures 1-3. All growth rates are recorded in microns per day, and represent measurements on first digits only, these being common to all subjects in the study group.

TABLE 1

Nail growth rates for various categories of subjects [1]

	Male					Female				
	Number	Mean	Standard Deviation	Coefficient of Variation	Range	Number	Mean	Standard Deviation	Coefficient of Variation	Range
Total [2]	179	117.0	16.6	14.4	74–168	121	111.9	17.2	15.4	74–155
Under 10	2	112.0	—	—	107–117	0				
10–29	149	119.6	15.3	12.8	84–168	80	116.6	16.7	14.3	79–155
30–39	12	114.8	9.7	8.4	99–132	17	103.2	16.6	16.1	74–145
40–49	9	101.1	7.5	7.4	88–117	12	103.3	10.5	10.2	86–122
50–69	7	88.9	9.4	10.6	74–102	5	101.2	13.2	13.0	84–120
Nail Biters	29	128.2	18.0	14.0	86–168	10	135.9	15.2	11.2	107–155
Non-Biters	113	117.4	13.7	11.7	84–155	59	115.2	14.8	12.8	91–147
Overweight [3]	15	116.3	20.3	17.5	84–168	8	124.3	11.9	9.6	109–145
Underweight [3]	16	125.5	16.7	13.3	91–155	15	117.0	17.1	14.6	94–152
Heavy [4]	15	121.3	13.2	10.9	94–137					
Light [5]	14	125.8	15.4	12.2	107–155					
Tall [6]	21	122.7	17.3	14.1	91–163					
Short [7]	21	119.0	18.9	15.9	84–168					
Weight Gaining						31	119.4	15.9	13.3	94–152
Weight Losing						13	122.2	20.7	16.9	94–155
Smokers [8]	25	111.1	14.6	13.1	84–155	11	113.3	10.5	9.3	102–130
Non-Smokers	71	120.7	14.1	11.7	89–168	36	116.4	14.9	12.8	89–145
Smokers, Non-Biters [8]	20	111.0	15.6	14.1	84–155	11	113.3	10.5	9.3	102–130
Non-Smokers, Non-Biters	55	117.3	12.1	10.3	89–145	33	114.7	14.1	12.3	89–145

[1] All figures in microns per day for first digits only. Except for the age data and totals, all data are from medical students and student nurses only and from within the age range 10–29.

[2] Includes three females whose specific ages were not provided.

[3] Outside the limit indicated as normal for height and sex in tables of the Metropolitan Life Insurace Company.

[4] Over 190 lbs.

[5] Under 150 lbs.

[6] Over 72 inches.

[7] Under 68 inches.

[8] Includes only those consuming 10 or more cigarettes per day.

The overall mean growth rate was 115 ± 17 microns per day, with a range of 74 to 168. The rate was relatively regular in most individuals, the average deviation (of single readings from each individuals's respective mean) being 5.5 ± 6.5 microns. There was con-

TABLE 2

Mean intra-individual variation in nail growth rate for various age and categorical groups [1]

Group	Sex	Number [2]	Mean	Standard Deviation	Coefficient Variation	Range [3]
Total	M&F	243–824	5.5	6.5	118.2	0–42
Total	M	149–439	4.3	4.5	104.7	0–19
Total	F	94–385	6.8	8.1	119.1	0–42
Under 10	M	2–10	10.2	6.9	67.6	3–12
10–29	M	123–329	3.6	3.7	102.8	0–16
30–39	M	11–56	7.3	6.5	89.0	2–14
40–49	M	7–26	4.3	3.8	88.4	0–6
50–69	M	6–18	4.9	4.0	81.6	2–8
10–29	F	66–281	6.4	8.2	128.1	0–42
30–39	F	13–50	5.6	5.7	101.8	0–10
40–49	F	9–24	9.8	10.1	103.1	2–37
50–69	F	6–30	9.8	6.7	68.4	3–13
Nail biters	M	20–54	4.5	3.4	75.7	10–93
Nail biters	F	8–32	6.2	7.4	119.4	25–80
Non-biters	M	99–261	3.5	3.5	100.0	0–16
Non-biters	F	50–209	6.1	8.4	137.7	0–42
Weight-gaining	F	22–109	7.3	10.5	69.4	3–42
Weight-losing	F	10–41	6.7	7.6	122.6	2–17
Overweight	M&F	20–65	4.4	5.1	115.8	0–10
Underweight	M&F	22–85	6.9	11.1	161.2	1–42
Smokers	M&F	30–88	4.3	4.2	97.7	1–9
Non-smokers	M&F	88–284	4.6	4.2	91.3	1–16

[1] All figures in microns per day, for first digits only.
[2] Includes all subjects with two or more readings. First number indicates total subjects; second indicates total readings.
[3] Indicates mean intra-individual variations for given subjects.

siderable variation between the hands, but no apparent relationship to so-called "handedness." Of 209 subjects in whom this comparison could be made satisfactorily, the nails on the right hand grew more rapidly in 37 (17.7%), and those on the left in 33 (15.8%). The hand rates were equal in 59 (28.2%) and variable (in different digits and/or at different times) in 80 instances (38.2%).

Of 136 subjects in whom the relative rates of nail growth could be satisfactorily determined, the rate was most rapid in the third digit in

[4] Includes only those consuming 10 or more cigarettes per day.

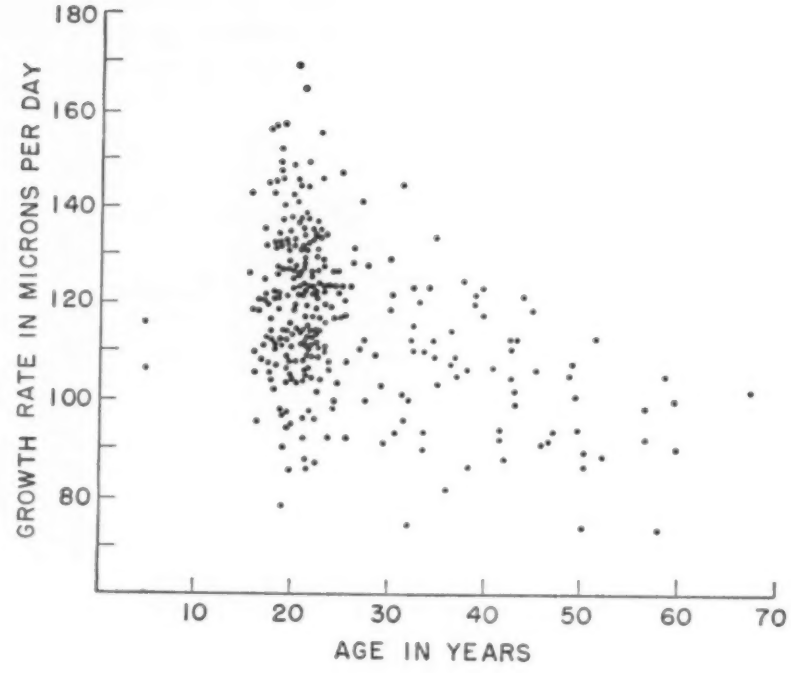


FIGURE 1.

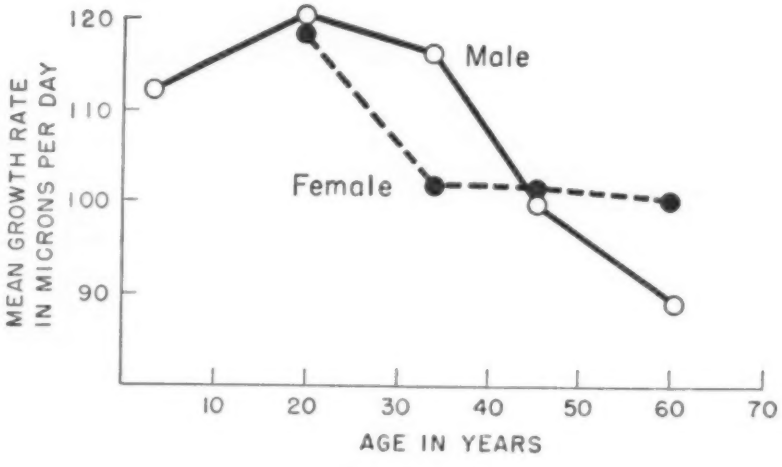


FIGURE 2.

91 (67%) and in the first in 16 instances (11.8%). The rates were identical in the first and third digits in 17 individuals (12.5%) and variable in 6 (4.4%). In 6 other persons growth was most rapid in one of the other digits.

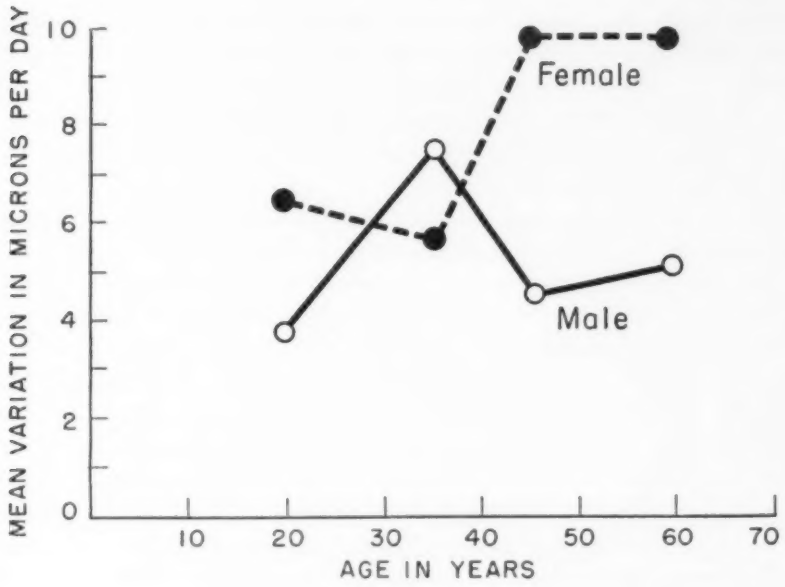


FIGURE 3.

In addition, the following general impressions were formed:

There was no seasonal variation in nail growth. The rate was unaffected by minor intercurrent illness, change in work or dietary habits, or by moderate stress situations (*e.g.*, scholastic examination periods). Subtotal thyroidectomy for adenoma in one individual did not appreciably modify growth, and the rates among the 7 patients did not differ significantly from those of ostensibly normal persons in the same age groups. Rates were similar in the identical and fraternal twins, although an older sibling (same sex) of the latter pair showed much more rapid nail growth.

More specifically, there was no statistically significant difference in average nail growth rate between tall and short, heavy and light or overweight and underweight individuals; nor between those gaining and those losing weight during the period of observation. Overweight

subjects exhibited less intra-individual variation than those below normal weight, as did those losing compared with those gaining weight, although these differences were not statistically significant.

The growth rate was significantly greater (P less than .001) among nail biters than among non-biters—so that other comparisons involved correcting for this factor.[1] For example, the crude growth rate among non-smokers was significantly greater (P less than .01) than among smokers, but the difference was not statistically significant after the biters had been eliminated from each group (possibly an effect of sample size in part, since each group included nearly the same proportion of individuals admitting this habit).

There was no apparent relationship of overall nail growth rate or variability to the subject's ordinate birth status, nor to his mother's age at the time of his birth. Likewise, there was no apparent relationship between these characteristics and individual life expectancy (as estimated from forebear experience), nor between them and a family history of cancer, hypertensive and coronary artery disease,[2] or diabetes mellitus.

There was no apparent difference between the nail growth rates of white and non-white subjects, but the latter group was too small for valid conclusions. The rate did, however, seem to relate to the factors of sex and age, being generally more rapid in males and in younger individuals. These variations are depicted in figure 2.

The mean growth rate for all males was 117 ± 17, with a range of 74 to 168 microns per day. For females it was 112 ± 17, with a range of 74 to 155. This difference was statistically significant (P less than .01). In the age group 10 to 30, representing a majority of all subjects, the average rates were 120 ± 17 and 115 ± 17 for males and females, respectively. This difference was not statistically significant. However, a significant fall (P less than .01) in the average female rate (to 103)

[1] The incidence of nail biting among overweights (34.8%) was significantly greater (P is less than .05) than among underweights (9.7%); it was also significantly greater (P less than .05) among those losing weight (46.2%) than among those gaining weight (16.1%) during the period of observation. Individuals with family histories of coronary artery disease likewise included a relatively high incidence of nail biters.

Other mechanical stress, *i. e.*, the regular application of nail polish and remover seemed not to effect nail growth rates.

[2] The mean intra-individual variation was not significantly increased among subjects with a family history of hypertension, but the ratio of individual mean variation to individual overall mean growth rate seemed highest in this group.

in the group 30 to 40 years of age, compared to only a slight contemporaneous decrease in the males, resulted in a significant difference (P less than .05) between the sexes in this decade. Successive, statistically significant drops in the male rates for the groups 40 to 50, and 50 to 70 years of age produced a mean of 89 among these last grouped subjects. This was substantially below the average of 101 for their female counterparts, although the gap was not statistically significant. The differences between mean growth rates for the groups under 40 and those over 40 were statistically significant (P less than .001) for each sex individually, and for both sexes considered together.

Inter-individual variation, as reflected in the coefficients of variation, was greater for females than males in all age groups.

Intra-individual variation, as represented by average variations from respective individual means, was likewise greater among females than among males (figure 3), but appeared to have no overall relationship to age. The mean variation for all males with two or more individual readings (4.3 ± 4.5) was significantly different (P less than .002) from that for all females (6.8 ± 8.1). It was also significantly lower in each age group, except in persons 30 to 40, among whom, unaccountably, the females exhibited less intra-individual variation. Again, the females generally showed more inter-individual differences, with greater coefficients of variation in all but the 50 and over age group.

COMMENT

In general these results are in accord with others previously reported, notably in respect to the average growth rate of about 0.1 millimeter per day, the lack of relationship to so-called handedness and the apparent propensity for more rapid growth of the nail of the third digit in most instances. The present observations, however, appear not to support the impression of seasonal growth changes, emphasized by Clark and Buxton ('38) and Gilchrist and Buxton ('39), but are in agreement, rather, with Bean's ('53) similar failure to demonstrate such a relationship during his 10 year self-study. They likewise seem consistent with his impression that the nail growth rate decreases with age and is essentially unaffected by changes in work habits, physical environment or by mild, intercurrent infection—although it does undeniably vary at times for reasons not yet identified. The considerable inter-individual variation evidenced in the present group is in accord with the large-scale observations of Clark, Buxton and Gilchrist, whose impressions of a lack

of consistent relationship between nail growth rate and age or sex may have been due to the relatively narrow span of early years represented by their two series. Berthold's contentions, as noted by Edwards ('38) and based on a wider age range of subjects, would seem to support further the concept of some relationship of nail growth to the period of life.

Intra-individual variation, in contradistinction to differences between persons, apparently has not been emphasized previously in the literature. In this study the wide variety of personal growth patterns observed among the subjects and the apparently real difference between the sexes noted in respect to constancy of rate, enhance the impression that these individual irregularities may ultimately prove of more biological significance than average nail growth rates.

The implications of these observations are not readily apparent—apart from a possible endocrine cyclic component operating in females (but evidently not limited to the premenopausal group). At present, individual nail growth rates seem not to be valid indices of personal health or to comprise a likely, practical means of detecting early deviations from the normal or satisfactory state of general wellbeing. However, the notable inter- and intra-individual variation may actually reflect fundamental metabolic differences, some conceivably at the "end organ" level, among normal functioning persons. The possibility that nail growth patterns may be related to other, more basic, biological phenomena governing the overall physical state and the future expectations of the individual warrants further investigation.

SUMMARY

Fingernail growth rates were measured in 300 individuals, including 293 ostensibly normal subjects and 7 with chronic disease.

The overall mean growth rate of 119 microns per day was consistent with impressions provided by other studies, although considerable intra- as well as inter-individual variation was observed, notably among females.

In general, nail growth seemed more rapid in the third digit, in males, in the younger age groups and in nail biters. It was questionably slower among smokers than non-smokers.

Nail growth rate seemed unrelated to such factors as season, handedness, body height and weight, minor changes in living habits and mild intercurrent illness. The 7 individuals with chronic disease showed no

significant difference in growth rate from their ostensibly healthy counterparts.

The explanation for these observed variations is not apparent, and further investigation of nail growth patterns is indicated—especially studies of intra-individual change—for possible basic biologic implications and practical clinical applications of these easily measured phenomena.

The author acknowledges the assistance of Miss Marie Leogrande who performed many of the fingernail measurements.

LITERATURE CITED

BEAN, W. B. 1953 A note on fingernail growth. J. Investig. Dermatol., *20*: 27-31.

CLARK, W. E. LEGROS AND L. H. D. BUXTON 1938 Studies in nail growth. Brit. J. Dermatol. and Syphil., *50*: 221-235.

EDWARDS, L. F. 1938 The rate of growth of finger nails in relation to their cystine content in arthritics. Ohio J. Science, *38*: 244-250.

GILCHRIST, M. L. AND L. H. D. BUXTON 1939 The relation of finger-nail growth to nutritional status. J. Anatomy, *75*: 575-582.

SHARPEY-SCHAFER, E. 1930 Observations on the relative growth of the nails on the right and left hands respectively: On seasonal variations in the rate, and on the influence of nerve section upon it. Proc. Roy. Soc. Edinburgh (Pt 1), *51*: 8-13.

SUNDERLAND, S. AND L. J. RAY 1952 Effect of denervation on nail growth. J. Neurol, Neurosurg. and Psychiat., *15*: 50-53.

WIGAND, R. 1937 Rate of growth of fingernails. Deutsche Ztschr. f. d. ges. gerichtl. Med., *29*: 75-80.

THE ABSENCE OF THE SICKLE CELL GENE IN SEVERAL MELANESIAN SOCIETIES AND ITS ANTHROPOLOGIC SIGNIFICANCE

BY DARIS R. SWINDLER *

Evans Institute of Dentistry and
Philadelphia Center for Research in Child Growth and Development

INTRODUCTION

WITHIN the last few years numerous papers have appeared concerning the sickling phenomenon (Pauling *et al.*, '49, Neel *et al.*, '51, Neel, '51b), the inheritance of the sickle cell gene (Beet, '49, Neel, '49, '51a, Lehmann, '51), and the distribution and ethnographic significance of this blood dyscrasia (Lehmann and Raper, '49, Hiernaux, '52, Foy *et al.*, '52). Perhaps the clearest statement is one by Singer ('53), in which he summarizes the information available on the sickle cell gene, as well as raises certain theoretical problems important to racial anthropology. The sickle cell (Neel's homozygous-heterozygous theory of inheritance is accepted in this paper) is widely distributed throughout Africa (Singer, '53) and has recently been reported among three Veddoid tribes of Central India (Lehmann and Cutbush, '52). The gene also has a sporadic frequency in other parts of the world where Negro admixture may have occurred (Caminopetros, '52, Dunlop and Mozumder, '52, Powell *et al.*, '50, Allison, '53). This information, coupled with a suggestion by Dr. Neil Tappen that the author check for sickling while in Melanesia, was responsible for the survey reported here.

* Physical anthropologist with an ethnographic expedition to New Britain jointly sponsored by the Museum and Department of Anthropology of the University of Pennsylvania. Additional funds were contributed by the American Philosophical Society and the Tri-Institutional Pacific Program. The author's participation was aided by a Wenner-Gren Foundation pre-doctoral fellowship.

The purpose of this paper is threefold: 1) to report the absence of the sickle cell gene among several Melanesian societies; 2) to consider the anthropologic implications that may be adduced with respect to the genetic affinities of the Oceanic Negro in the light of this new evidence; and 3) to discuss what significance the sickle cell gene affords the student of race in view of the most recent hypotheses about this abnormal blood characteristic.

MATERIALS AND METHODS

Since some doubt has been registered about the reliability of the techniques and results of several investigations (see especially Singer, '53), the author spent two weeks before departing for Melanesia in the Department of Hematology at the Children's Hospital of Philadelphia studying the sickling phenomenon. During this period he had the opportunity to observe blood samples from individuals with the sickle cell trait and sickle cell anemia. He was also taught a simple and reliable technique for eliciting sickling in which a reducing agent, sodium metabisulfite, is employed.

The field method consisted of dissolving a three-grain tablet of sodium metabisulfite in approximately 10 cc of sterile water. Fresh preparations were made daily to ensure maximum strength of the solution. Recently, Allison ('54a) has advocated this procedure when using chemical reducing agents in the tropics. One drop of blood was obtained from each individual by finger prick and placed on a glass microscope slide. Immediately a drop of metabisulfite solution was added to the slide and mixed thoroughly. A coverslip was then placed over the preparation and ringed with a thin layer of petroleum jelly, which was melted with a match flame and then permitted to solidify. The slide was then ready for microscopic study at various intervals. All slides were checked for sickling at 15 minutes, 24 hours, 48 hours and, when feasible, at 72 hours, although in a few instances the blood had deteriorated and the erythrocytes were not discernible after 72 hours. Consequently, if no sickling had occurred by the 48-hour check the individual was considered negative for drepanocytosis (sickling).

Blood smears were also made for each individual. The cells were preserved by pouring methyl alcohol over the slide. These preparations were returned to the Children's Hospital, where Dr. Patraula Typaldou examined them for sickle cells and other blood features. This study was conducted six months after the smears were collected.

A total of 277 blood samples were collected and studied in the field.

Of these, 156 were from the West Nakanai, who live along the Hoskin's Peninsula, which is located on the north central coast of New Britain in the Territory of Papua and New Guinea. The remaining 121 slides were collected and examined at a small hospital unit in Talesea, which is a government outpost situated about 75 miles northwest of the West Nakanai area. Here we had the opportunity to investigate members of various tribes in the vicinity when they came into Talasea for medical attention. In fact, the series include three "police boys" from the Sepik River regions on New Guinea, as well as natives from Vitu and

TABLE 1

Individuals separated as to tribe, age, and sex

	ADULT		CHILDREN	
TRIBE	MALE	FEMALE	MALE	FEMALE
West Nakanai	99	44	7	6
Bakovi	28	11	5	1
Kambi	23	15		
Bulu	8	6	2	1
East Nakanai	2	2	2	
Central Nakanai	1			
Taulai (Rabaul)	2		1	
Banale	1			
New Guinea (Sepik R.)	3			
Bali Island	1		2	1
Vitu Island	2		1	
Total	170	78	20	9

Bali islands which lie off the north coast of New Britain. We were quite particular to exclude from both series all near relatives; in other words, not more than one member of a family was included in the survey.

The separation of both series into tribe, sex, and age categories may be seen in table 1. The division between adults and children is somewhat arbitrary since chronologic age is not recorded. We could only estimate the individual's maturational status as registered by his or her acquisition of certain well-known secondary sexual characteristics plus Hellman's dental ages. In short, if an individual lacked any indication of axillary hair or beard and his third molar teeth were unerupted, he was classified as a child.

RESULTS AND DISCUSSION

There was no indication of sickle cell anemia or sickle cell trait in the slides examined by us in the field or in the returned smears studied by Dr. Typaldou. Unfortunately the sample is rather small, but we feel justified in stating that at least among one Melanesian society, the West Nakani, the sickle cell gene is absent.

Before considering the anthropologic implications of this finding, attention will be directed to other studies which have been conducted in this general area for the sickle cell, and which appear to corroborate our data. During a disease survey of the Malayan aborigines, Polunin ('52) did not find sickling to be present in a group of 150 Malayan Negritos. Sneath ('54) reports the absence of the sickle cell in both the Malayan Negritos and aboriginal Malays. His samples consisted of about 100 members of each group. According to Horsfall and Lehmann ('53) there was no evidence of sickling among 52 Onges of the Little Andaman Islands and 16 Andamanese of mixed Negrito ancestry studied by Lehmann. They also report the absence of the sickle cell gene in 72 Australian aborigines. Of these, 57 individuals were of unmixed Australian ancestry and presented many Carpentarian (Veddoid) characteristics. Simmons *et al.*, ('53) also failed to find any evidence of the sickle cell in 125 random adult blood samples obtained from Western Australia. These data, coupled with the results presented herein, strongly suggest that the sickle cell gene, wherever its origin or origins may have been, did not pass any farther eastward than India.

Of the many racial elements represented throughout Melanesia today the influence of the Oceanic Negrito and Negroid is the most widely disseminated. The Mongoloid and Caucasoid contributions are also present in various parts of the area but are of little consequence to the hypotheses of the present paper. These 4 components then form the matrix of the racially blended populations inhabiting New Guinea and the remainder of Melanesia. The origins of the Oceanic Negrito and Negroid are shrouded in antiquity and will probably never be completely unraveled. Most authorities would agree, however, that they are genetically related to their African counterparts, the African Pygmy and Negro. If this relationship can be proven and if the sickle cell gene is actually indicative of Negro ancestry it is incumbent upon us to elucidate the enigma. Birdsell ('49, p. 3) is most emphatic concerning the origin of the Oceanic Negrito; he writes, "If there is any safe inference in the field of racial anthropology, it is the Oceanic Negritos

must be considered as very closely related genetically to the Negritos of the Congo Basin in tropical Africa." Earlier, Howells ('43, p. 43) was somewhat less absolute in his opinion, but nevertheless stated that "It is very difficult to doubt that the pygmy peoples of New Guinea are fundamentally of the general Negrito stock." In the second edition of *Introduction to Physical Anthropology* Montagu ('51, p. 315) concurs rather ambivalently with this idea, for he writes, "The superficial resemblances of the pygmies have inclined many students to consider them as closely related. Their peculiar distribution would tend to support this view." The consensus, then, is that these two groups are genetically related, but differences of opinion arise with respect to the degree of genetic affinity between them.

When the Oceanic Negro is considered, his relationship to the African Negro is more ambiguous. Here there is a definite dichotomy. Some scholars maintain that there is a full-sized Negroid element in Melanesia which may have a very ancient origin in Asia (Howells, '43, Montagu, '51). Birdsell ('49, p. 6) does not agree with this thesis but states, "I am confident that any so-called Negroid, as opposed to Negritoid type, in New Guinea and Melanesia results from hybridization of Negrito and Carpentarian [Veddoid] elements present throughout much of the archipelago." Whatever the ultimate outcome, it seems legitimate to assume that "some" genetic relationship exists between the African and Melanesian populations.

It has been shown that there is a probable genetic link between Africa, Australasia, and Melanesia. Why then, is the sickle cell gene not present in the last two regions? To answer this, one must determine if the presence of the sickle cell gene in a population is actually indicative of Negro ancestry. At the present time the answer to this question is not satisfactorily known. There are those who maintain that the sickling feature of the erythrocytes cannot be regarded as a peculiarity of the Negro people, and its ethnological significance may have been overrated in the past (Foy *et al.*, '54, '55). In 1952 Lehmann and Cutbush ('52, p. 404) were of the opposite opinion and wrote, "So far as we know, the sickle cell trait and sickle cell anemia have not been reported in people who were not either Africans or members of groups which must be expected to have an admixture of African blood." Lehmann ('53, p. 9), however appears to have shifted his allegiance, for he later wrote, "The trait is not a universal African feature and one may suggest that it entered the continent with Veddian blood well after the African races

had established themselves." He also says that the gene cannot be used as a "tracer" of African ancestry as is the case with the R^o (cDe) chromosome. An interesting suggestion made by von Haimendorf ('54) is apropos here, irrespective of its validity. He believes the introduction of the sickle cell into the blood of the Todas of the Nilgiris Hills of India can perhaps be explained by miscegenation. In 1953 he discovered a hitherto unnoticed system of wife-lending between the Todas and the Mudugas of Pottikal. Certain village headmen of these two groups have traditional friends who visit them and the host is expected to vacate his home, but the wife remains and the visitor assumes the role of the husband. The discovery of this system of hereditary friendships led von Haimendorf ('54, p. 29) to suggest that "the slight incidence of the sickle-cell trait in the blood of Todas can perhaps be explained by such miscegenation . . . and may thus help in the solution of a genetic puzzle." The awareness of such sociological systems may help explain the distribution of a trait among supposedly endogamous groups, but in the present case, it is unknown whether the Mudugas possess the sickle cell gene. (See list of Veddoid tribes examined to date in Lehmann and Cutbush, '52). Thus the verity of von Haimendorf's statement can only be established by further field work. However, such cultural phenomena are too often overlooked by the physical anthropologist unfamiliar with the mores of the people he is studying. Singer ('53, '54), on the other hand, staunchly maintains that the sickle cell gene is found exclusively in Negroes or in persons who have had Negro admixture; in fact, he cites Central Africa as the probable home of the gene. About all one can say at this time is that the presence of the sickle cell in a population is not unequivocal evidence of Negro ancestry. However, irrespective of whether the sickle cell is peculiar to Africans (Singer, '54) or arose among some pre-Veddoid group (Lehmann and Cutbush, '52, Mourant, '52), one must attempt to explain its absence in Melanesia and Australasia because genes from one or the other of these racial elements are supposedly present in this area.

Within the last few years it has been almost conclusively proven that the sickle cell trait (heterozygous condition) has a selective value in a malarious environment (Beet, '46, Choremos *et al.*, '51, Raper, '54, and Allison, '54a, '54b, and '54c). Although this hypothesis is not without its antagonists (notably Foy *et al.*, '55, Moore *et al.*, '54), it is beginning to gain favor, especially since the revealing work by Allison and Raper. These investigators suggest that the heterozygote is favored by a high

degree of resistance to malarial parasites, especially with respect to *Plasmodium falciparum*. According to Allison ('54a) *P. falciparum* is the least effective of the malarial parasites since it is not found as frequently as other forms in infected individuals. He further states ('54b, p. 317), "There is little sickle cell south of the Zambesi River because it is not as malarious plus the fact *P. falciparum* is being replaced to some extent by *P. vivax* as the dominant species." It appears that sicklers (heterozygotes) may be naturally resistant to subtertian malaria so that they have a selective advantage over the normal homozygotes in regions where malaria is endemic. In other words, balanced polymorphism may be the explanation for the maintenance of the sickle cell gene. If so, it seems only logical to conclude that the gene had a relatively recent origin, since it is well known that malaria is endemic in most regions of Australasia and Melanesia. By recent I simply mean after the major differentiation and establishment of the populations in those areas, which was undoubtedly sometime during the last half of the fourth glacial period. This suggestion is essentially in agreement with those proposed by Singer ('53), Lehmann ('52), and Mourant ('54).

Another factor which one cannot overlook is genetic drift. If this phenomenon has been operating, present sickling rates in a tribe may not bear much relationship to the frequency of the gene when originally introduced, and individual tribal frequencies would have limited ethnologic significance (Brues, '55). Foy *et al.* ('54) believe that this is the situation which obtains in Africa and that genetic drift, rather than natural selection, is the cause of wide inter-tribal and intra-tribal variability. However, before one too freely entertains the concept of genetic drift to explain all variability, one should recall the criticisms by Glass *et al.* ('52) on such uncritical application of this evolutionary mechanism. With the information now available, balanced polymorphism seems much easier to validate scientifically, although it is quite possible that both phenomena are operating simultaneously on the gene.

CONCLUSION

The previous discussion has attempted to elucidate some of the anthropologic problems concerning the sickle cell gene. It appears that the gene has limited ethnologic application and that its utilization as a "tracer" of Negro ancestry is open to suspicion. The suggestion has been made that the gene never passed eastward of India and that this is a consequence of its relatively recent acquisition in *Homo*. This hypo-

thesis seems even more tenable in view of the data presented here. It is demonstrated that the absence of the gene in Australasia and Melanesia does not substantially alter previous concepts on the origin of these populations.

ACKNOWLEDGMENTS

I am deeply grateful to Dr. J. Irving Wolman, Director of the Department of Hematology at the Children's Hospital of Philadelphia, his staff, and especially Dr. Patroula Typaldou for their generous assistance. Acknowledgment is due to the Medical Officer-in-Charge of the Talasea Hospital, Mr. William Bell, for granting the facilities required.

LITERATURE CITED

ALLISON, A. C. 1953 The sickle-cell trait in the Mediterranean area. Man, *53*: 23-24.

——— 1954a Protection afforded by sickle-cell trait against subtertian malarial infection. Brit. Med. J., *1*: 290-294.

——— 1954b The distribution of the sickle-cell trait in East Africa and elsewhere, and its apparent relationship to the incidence of subtertian malaria. Trans. Roy. Soc. Trop. Med. Hyg., *48*: 312-318.

——— 1954c Notes on sickle-cell polymorphism. With statistical appendix by S. M. Smith. Annals of Human Genetics, *19* (Part 1): 39-57.

BEET, E. A. 1946 Sickle-cell disease in the Balovale district of Northern Rhodesia. East Afr. Med. J., *23*: 75-86.

——— 1949 The genetics of the sickle-cell trait in a Bantu tribe. Ann. Eugen., *14*: 279-284.

BIRDSELL, J. B. 1949 The problem of the early peopling of the Americas as viewed from Asia. Papers on the Physical Anthropology of the American Indian. The Viking Fund Inc., New York, 1-86.

BRUES, A. M. 1954 Selection and polymorphism in the A-B-O blood groups. Am. J. Phys. Anthrop., n. s., *12*: 559-597.

CAMINOPETROS, J. 1952 The sickle-cell anomaly as a sign of Mediterranean anemia. Lancet, *262*: 682-693.

CHOREMIS, C., N. ZERVOS, V. CONSTANTINIDES, AND L. ZANNOS 1951 Sickle-cell anaemia in Greece. Lancet, *260*: 1147-1149.

DUNLOP, K. J., AND V. K. MOZUMDER 1952 The occurrence of sickle-cell anemia among a group of tea garden labourers in Upper Assam. Indian Med. Gaz., *87*: 387-391.

FOY, H., A. KONDI, AND A. HARGREAVES 1952 Anaemias of Africans. Trans. Roy. Soc. Trop. Med. Hyg., *46*: 327-358.

FOY, H., A. KONDI, G. L. TIMMS, W. BRASS AND F. BUSHRA 1954 The vari-

ability of sickle-cell rates in the tribes of Kenya and the southern Sudan. Brit. Med. J., *1*: 294-297.

FOY, H., W. BRASS, G. L. TIMMS AND R. A. MOORE 1955 Sickling and malaria. Brit. Med. J., *1*: 285-286.

GLASS, B., M. S. SACKS, E. F. JAHN AND C. HESS 1952 Genetic drift in a religious isolate: an analysis of the causes of variation in blood group and other gene frequencies in a small population. Amer. Nat., *86*: 145-159.

HIERNAUX, J. 1952 La génétique de la sicklémie et l'intérêt anthropologique de sa fréquence en Afrique noire. Annales du musée Royal du Congo Belge Tervuren (Belgique). Sciences de l'Homme. Anthropologie. *2*: 7-42.

HORSFALL, W. R., AND H. LEHMANN 1953 Absence of the sickle-cell trait in 72 Australian aboriginals. Nature, *172*: 638.

HOWELLS, W. W. 1943 The racial elements of Melanesia. Studies in the Anthropology of Oceania and Asia., C. C. Coon and J. M. Andrews, eds. Papers of the Peabody Museum of American Archaeology and Ethnology, Cambridge, Mass. *20*: 38-49.

LEHMANN, H. 1951 Sickle-cell anemia and sickle-cell trait as homozygous and heterozygous gene combinations. Nature, *167*: 931-933.

——— 1953 The sickle-cell trait: not an essentially Negroid feature. Man, *53*: 9.

LEHMANN, H. AND M. CUTBUSH 1952 Sickle-cell trait in southern India. Brit. Med. J., *1*: 404-405.

LEHMANN, H. AND A. B. RAPER 1949 Distribution of the sickle-cell trait in Uganda, and its ethnological significance. Nature, *164*: 494-495.

MONTAGU, M. F. ASHLEY 1951 An Introduction to Physical Anthropology, 2nd ed. Charles C. Thomas, Springfield, Ill.

MOORE, R. A., W. BRASS AND H. FOY 1954 Sickling and malaria. Brit. Med. J., *1*: 630-631.

MOURANT, A. E. 1952 The sickle-cell trait. Editorial. Brit. Med. J., *1*: 426-427.

——— 1954 The Distribution of the Human Blood Groups. 1st ed. Charles C. Thomas, Springfield, Ill.

NEEL, J. V. 1949 The inheritance of sickle-cell anemia. Science, *110*: 64-66.

——— 1951a The inheritance of the sickling phenomenon with particular reference to sickle-cell disease. Blood, *6*: 389-412.

——— 1951b Perspectives in the genetics of sickle-cell diseases. Blood, 7: 467-472.

NEEL, J. V., I. C. WELLS AND H. A. ITANO 1951 Familial differences in the proportion of abnormal hemoglobin present in the sickle-cell trait. J. Clin. Invest., *30*: 1120-1124.

PAULING, L., H. A. ITANO, S. J. SINGER AND I. C. WELLS 1949 Sickle-cell anemia, a molecular disease. Science, *110*: 543-548.

POLUNIN, I. 1952 Anthropological problems encountered during a disease survey of Malayan aborigines. Man, *52*: 70-71.

POWELL, W. N., J. G. RODARTE AND J. V. NEEL 1950 The occurrence in a family

of Sicilian ancestry of the traits for both sickling and thalassemia. Blood, *5*: 887-897.

RAPER, A. B. 1954 Sickling and malaria. Brit. Med., J., *1*: 1162-1163.

SIMMONS, R. T., J. J. GRAYDON AND J. B. BIRDSELL 1953 High Rz frequency in the blood of the Australian aborigines. Nature, *172*: 500.

SINGER, R. 1953 The sickle-cell trait in Africa. Am. Anthrop., *55*: 634-648.

——— 1954 The origin of the sickle-cell. So. Afr. J. Sci., *50*: 287-291.

SNEATH, P. H. A. 1954 Anthropological blood grouping in southeastern Asia. Man, *54*: 28.

VON FURER-HAIMENDORF, C. 1954 Hereditary friendships and inter-tribal sex relations between Todas and Mudugas. Man, *54*: 28-29.

MORTALITY OF PERSONS WITH XYLOKETOSURIA

A FOLLOW-UP STUDY OF A RARE METABOLIC ANOMALY

BY MARGARET LASKER

Yonkers, New York

IT has long been known that xyloketosuria (a condition in which the pentose xyloketose occurs in human urine) is a recessively inherited anomaly which occurs in persons of all age groups (Lasker, Enklewitz and Lasker, '36). Mortality statistics are usually lacking for such anomalies, especially if they are rare or if they can be diagnosed only by special tests. Seventy-two cases of xyloketosuria have been followed, over half of them for 16 years or more. In one case an indirect follow-up of a patient diagnosed as pentosuric in Germany in 1913 was made possible by subsequent independent republication of the same case in South Africa 33 years later (Bloomberg, Alldis and Epstein, '46, and Lasker, '50) and has now been followed for 41 years. In 57 of the cases, I personally tested the urine and established that xyloketose was present. Several pentosurics were tested daily for months at a time, others repeatedly during the long follow-up periods. In 37 of the cases I retested within the last few years, I have found xyloketose present every time and always in a concentration usual for the particular individual. The total follow-up experience is 1043 person-years, an average of 14.5 years per individual.

Of the 72 persons with xyloketosuria, 12 (16.7%) have died. To determine whether this number is significantly more than would have occurred in non-xyloketosuric persons, the data have been compared with life tables. The mid-date between the year of diagnosis and the last year of follow-up was taken for each subject. Those for all the subjects were averaged and the mean falls in 1943. It is therefore appropriate to use data based on the census of 1940 and deaths recorded in the years 1939-1941 (State and Regional Life Tables 1939-1941).

FINDINGS

Table 1 shows the age at which each xyloketosuric case was first identified as one of essential pentosuria and the number of years it was followed. To determine the general white mortality in the United States for persons alive during the years of age corresponding to those of each of the cases, the Life Tables for whites in the United States were consulted. As a control for each case, the entry for the appropriate sex under "Number living at beginning of year of age" for the last age of contact with the pentosuric subject was subtracted from the corresponding entry for the age at first diagnosis, and the difference was divided by the former (State and Regional Life Tables 1939-41, pp. 10-13). The rate of mortality expected in persons between these ages is thus calculated from the table. In the case of persons who died before 1951, follow-up statistics were calculated on the basis of this date, which is the mean termination date of all the other follow-up studies. This is done on the assumption that these individuals, had they lived, could have been studied as easily as the others. The 72 duration-weighted rates were then added to give the total number of deaths expected in a control population of the same age- and sex-distribution. Approximately 8.9 of the 72 (12.4%) would be expected to die in the periods covered.[1]

The question arises whether the difference between the 12 deaths of xyloketosurics and the 8.9 deaths which would have been expected in a corresponding population sample is significant or meaningful. It is neither. Statistically a simple probability test cannot be used because the various cases contribute so unequally to the total experience. But even assuming an "n" of 72, however, the standard error ($\sqrt{\frac{pq}{n}}$) of 16.7% (our percentage mortality) would be $\sqrt{\frac{16.7\% \times 83.3\%}{72}} = \pm 4.4\%$. The difference between the observed and expected mortality ($16.7 - 12.4 = 4.3\%$) would thus be less than the standard error, hence not signifi-

[1] Three cases which were followed for a year or less have been excluded from consideration because of the shortness of the time they were followed. Had they been included, the mean date of termination of the follow-up periods for surviving individuals would have been less recent and the calculated potential follow-up of those who died would be shorter, hence the calculated expected number of deaths would be somewhat less. On the other hand, the one case in which a subject died within a year of the first diagnosis has been retained in the calculations.

TABLE 1

Follow-up Cases of Xyloketosuria

CASE	AGE PENTOSE IDENTIFIED	FOLLOM-UP PERIOD (YEARS)	YEAR OF LAST CONTACT
Males			
HS	2	3	1955
SB	3	8	1938
LD	3	3	1953
RP	3	4	1955
ME	4	16	1955
AB	4	15	1954
RB	5	41	1954
G	5	7	1953
BC	6	18	1953
MG	6	20	1955
IB	7	8	1955
DF	9	11	1954
HT	10	17	Died 1949
HZ	10	15	1955
RB	11	3	1938
LG	16	2	1938
MB	17	16	1955
L	19	14	1938
K	21	14	1938
JL	22	23	1955
DM	22	14	1952
CS	23	24	1955
LG	24	20	1955
NI	25	19	1955
BL	28	23	1955
HG	28	20	1955
KM	28	11	Died 1948
BR	29	5	1938
LG	30	20	1955
EB	31	20	1955
SS	31	17	1955
NL	33	22	1955
BR	33	1	Died 1934
MD	34	16	1955
JA	35	21	1953
LB	37	12	Died 1944
JD	38	13	Died 1951
BB	39	6	1939
LL	41	23	1954
SB	46	3	1955
ALW	48	30	Died 1938
JS	49	16	Died 1951
F	53	14	1938
AB	57	16	1955
SM	61	9	Died 1946

TABLE 1 (Continued)

Follow-up Cases of Xyloketosuria

CASE	AGE PENTOSE IDENTIFIED	FOLLOM-UP PERIOD (YEARS)	YEAR OF LAST CONTACT
Females			
L	6	3	1953
FD	7	17	1954
RC	8	2	1937
BS	8	6	1955
HD	9	17	1954
HG	9	17	1955
SJ	10	22	1955
JE	11	34	1952
FB	16	3	1938
BM	19	20	1955
EW	19	16	1955
RG	21	16	1955
MKG	23	21	1954
MC	27	21	1953
YZ	29	23	1955
SR	35	15	1952
CJ	37	22	1955
LB	40	12	1953
JG	40	20	1955
AG	41	23	1955
ES	42	3	Died 1939
RY	42	2	1955
RN	51	11	1952
EL	54	12	1945
BL	61	8	Died 1941
FM	67	18	Died 1953
PS	72	6	Died 1944

cant. Actually, the effective "n" is smaller; therefore the standard error is larger, and the difference even less significant. Also, I have used conservative assumptions: 1) For the general population I have used tables which apply to the actual age, that is, presumably about a half year below the average (and hence with lower mortality) than the xyloketosurics, whose ages were recorded as on the last birthday. 2) Although half of the cases came from the State of New York, I

have used total United States mortality statistics as a control; the death rates for the State of New York at these ages are appreciably higher. 3) General population mortality statistics were used although the diagnosis of xyloketosuria requires hospital laboratory tests. About 20 of the cases were first discovered in persons hospitalized for some reason unconnected with the sugar in the urine; for example, in one case the patient was in the hospital gravely ill with miliary tuberculosis at the time the xyloketose was identified in the urine, and died of tuberculosis within the year.

DISCUSSION

There is no evidence of an appreciably higher mortality in xyloketosurics than in the general white population of the United States. What little excess of deaths there may be in the former stems from a slight

TABLE 2

Deaths of Xyloketosurics

CASE	AGE AT DEATH	ASCRIBED CAUSE OF DEATH
Male		
HT	27	Bronchial disease
BR	35	Miliary tuberculosis
KM	39	Polycystic kidney disease
LB	49	Heart disease
JD	51	Coronary thrombosis
JS	65	Coronary thrombosis
SM	70	Unknown
ALW	78	Unknown
Female		
ES	45	Cancer of pancreas
BL	69	Unknown
PS	78	Unknown
FM	85	Unknown

excess of deaths in males 51 years old or less which may be a result of chance (table 2). It may also be seen in table 2 that, in so far as is known, death in xyloketosurics occurs from a range of causes similar to what one might expect in a general population of this age distribution.

As xyloketosuria is inherited, it may be asked whether the mortality experience can cast any light on the population genetics of this charac-

teristic. As I have said elsewhere (Lasker, '52), the condition seems to be virtually limited to Jews, and the geographical distribution suggests an increase in the gene frequency among them during the last thousand or two thousand years. Such an increase could result from any of three causes: a repeated mutation; some advantage for lower mortality or higher fertility conferred by the gene; or sheer chance factors in a relatively small population. Repeated mutation in a single population would still leave unexplained the reason for a higher mutation rate in one segment of the species but not in other genetically similar peoples; thus in the absence of any direct evidence favoring it, this hypothesis is not helpful.

The present data show no decreased mortality rate for xyloketosurics, but the total mortality experience is too small to permit an answer as to whether this might not be true for the critical years prior to and including the reproductive ages. In any case, for each homozygote, there must be many heterozygotic carriers of xyloketosuria; the mortality and fertility of these would therefore be more important in determining whether there is any "natural selection" favoring a rapid increase in the frequency of the gene for xyloketosuria. Unfortunately, no clinical test is known that will distinguish these carriers. If no factor pointing to natural selection is found, it may well be that in a small isolated population pure chance ("random genetic drift") may have contributed decisively to the present ethnic distribution of this genetic condition.

SUMMARY

In follow-up studies of 72 cases of xyloketosuria, 12 (16.7%) were found to have died. Among white persons of corresponding ages in the United States the death rate is 12.4%. The disparity is not statistically significant. Furthermore, this difference may result from the fact that many of the cases became known only because they were receiving clinical attention for other reasons. Even if the difference were found in much more extensive data—and these would be difficult to collect for such a rare metabolic anomaly—it might be accounted for by the conservative assumptions made in the calculations of the control. As cases are usually found by special clinical tests, some of the xyloketosurics who died may have been detected, and so were included in this study only because they had sought medical advice for illnesses—unrelated to xyloketosuria—which later caused, or contributed toward, their deaths. On the other

hand, the absence of evidence for a lower mortality of xyloketosurics leaves open the question whether favorable mortality and fertility rates (especially among heterozygotic carriers) or sheer chance factors better explain the present distribution of the condition.

LITERATURE CITED

BLOOMBERG, B. M., D. ALLDIS AND M. EPSTEIN 1946 Chronic essential pentosuria, with report of a case. So. African Med. J., May 25, 1-4.

LASKER, MARGARET 1950 The question of arabinosuria. Amer. J. Clin. Path., *20*: 485-488.

——— 1952 The geographic distribution of heterozygotes for essential pentosuria. Amer. J. Phys, Anthrop., n. s., *10*: 258-259 (abstract).

LASKER, MARGARET, M. ENKLEWITZ AND G. W. LASKER 1936 The inheritance of l-xyloketosuria (essential pentosuria). Human Biology, *8*: 243-255.

STATE AND REGIONAL LIFE TABLES 1939-41 [1948? No publication date given.] Federal Security Agency (Public Health Service, National Office of Vital Statistics, U. S. Government Printing Office, Washington, D. C.

A THEORY OF CULTURAL GENETICS *

BY BURT W. AGINSKY

The City College, New York City

INTRODUCTION

THE core of this presentation may be termed "A Theory of Cultural Genetics" because there are reports in anthropological literature of a variety of human genetic systems which are culturally recognized within each society. Whereas lower forms of animal life ignore or soon forget genetic relationships, human beings maintain their biological relationships through the use of language and records. A particular genetic system is traditionally called a kinship system. The kinship system seldom includes a recognition of all the genetic relationships of the individuals who make up the population, but rather some portion of it which is systematized and known to the people. The people participate upon that basis and thus their daily lives are affected in regard to inheritance, residence after marriage, social relationships of avoidance, reciprocity, respect, joking relations, pre-marital sex relations, and actual marriages. In short, participation in the culture has a direct effect upon the genetics.

The study of human genetics need not be limited to people of a particular time or place. The theory presented herein pertains to the variety of human genetic systems resulting from the reported cultural forms of marriage at many times and places.

Generally speaking marriage is considered the beginning of genetics under consideration. However, no matter which kind of marriage occurs,

* This is an abbreviated presentation of the results of research, experimentation, and testing which began in 1932. Summaries of this paper were presented at the meetings of the American Association of Physical Anthropologists of 1954 and 1955. Several previous publications by the author relate to this subject. (Aginsky, '35A, '35B, '39, '40)

there is no genetic resultant until children are present. Furthermore if one member of a marriage has a child with a nonmember of that marriage the geneticist is interested in that child and its biological parents rather than the cultural parents. Therefore, it is essential to differentiate between marriage which is cultural, and sexual mating which is genetic.

Sororate, levirate, or any other form of marriage, by itself or in combination, when standardized in a population, is of no significance unless children result. When, however, a standardized form of marriage is participated in by the population and is repetitive, then the effects upon the genetics of that population may be of interest to the geneticist.

THE 32 NUCLEAR STRUCTURES

The reported forms of marriage have been brought together without regard to time or space (place). Some of them are: monogamy, polyandry, ploygyny, group, double, exchange, sororate, levirate, mother's brother's wife, husband's sister's son, wife's brother's daughter, father's sister's husband, brother's daughter's husband, cross-cousin, grandparent-grandchild, and brother-sister. Each of these has been reported as being practiced in some societies as the sole form and in others in combinations.

There are 6 basic forms of marriage (see figure 1)

1′P *The Primary* (our own form of sexual mating may serve as an example)

The Secondaries

2′S Sororate
3′L Levirate
5′W Wife's brother's daughter
9′H Husband's sister's son
1″E Exchange (Double Primary)

▲ Man
● Woman
Marriage (sexual mating)
Husband and wife (who are parents)
The horizontal line represents siblings (in this case a brother and a sister)
The perpendicular line represents parent-child relationship. (Here we have a brother and sister of the same parents.)

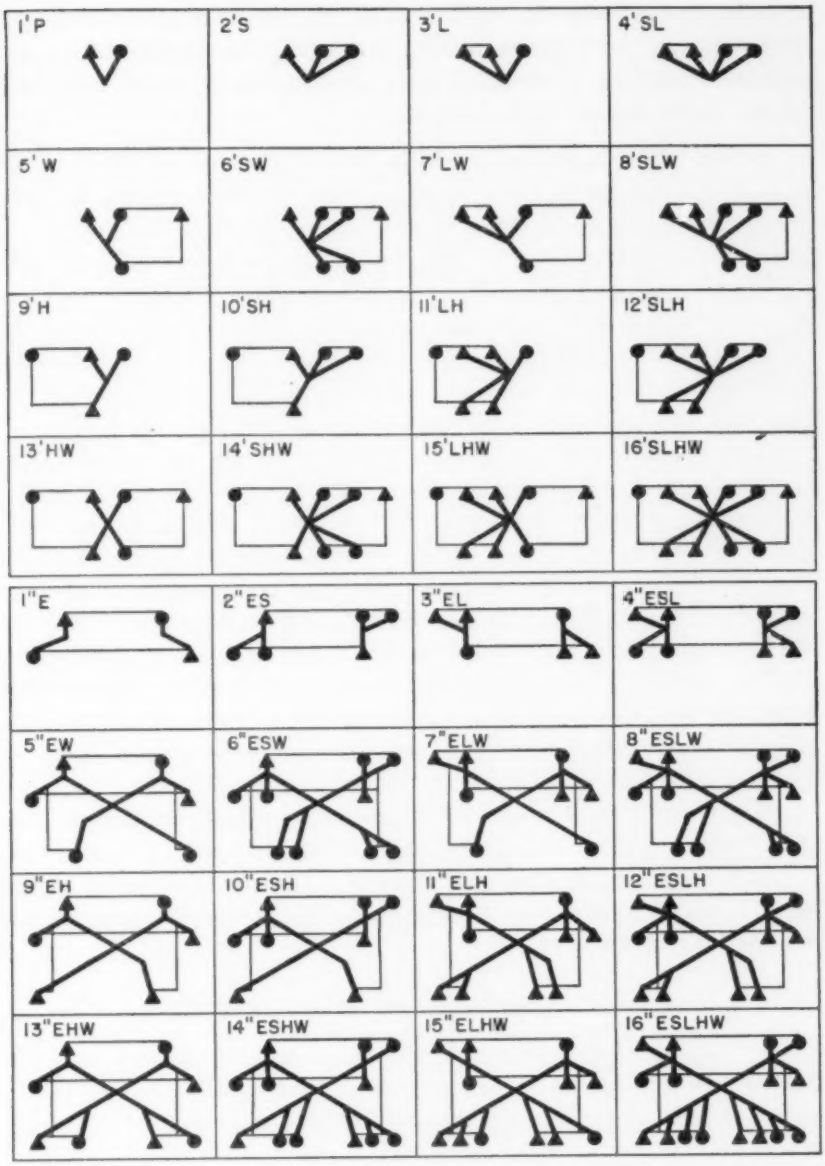


FIG. 1. PRIME AND DOUBLE PRIME SERIES.

there is no genetic resultant until children are present. Furthermore if one member of a marriage has a child with a nonmember of that marriage the geneticist is interested in that child and its biological parents rather than the cultural parents. Therefore, it is essential to differentiate between marriage which is cultural, and sexual mating which is genetic.

Sororate, levirate, or any other form of marriage, by itself or in combination, when standardized in a population, is of no significance unless children result. When, however, a standardized form of marriage is participated in by the population and is repetitive, then the effects upon the genetics of that population may be of interest to the geneticist.

THE 32 NUCLEAR STRUCTURES

The reported forms of marriage have been brought together without regard to time or space (place). Some of them are: monogamy, polyandry, ploygyny, group, double, exchange, sororate, levirate, mother's brother's wife, husband's sister's son, wife's brother's daughter, father's sister's husband, brother's daughter's husband, cross-cousin, grandparent-grandchild, and brother-sister. Each of these has been reported as being practiced in some societies as the sole form and in others in combinations.

There are 6 basic forms of marriage (see figure 1)

1′P *The Primary* (our own form of sexual mating may serve as an example)

The Secondaries

2′S Sororate
3′L Levirate
5′W Wife's brother's daughter
9′H Husband's sister's son
1″E Exchange (Double Primary)

▲ Man
● Woman
Marriage (sexual mating)
Husband and wife (who are parents)
The horizontal line represents siblings (in this case a brother and a sister)
The perpendicular line represents parent-child relationship. (Here we have a brother and sister of the same parents.)

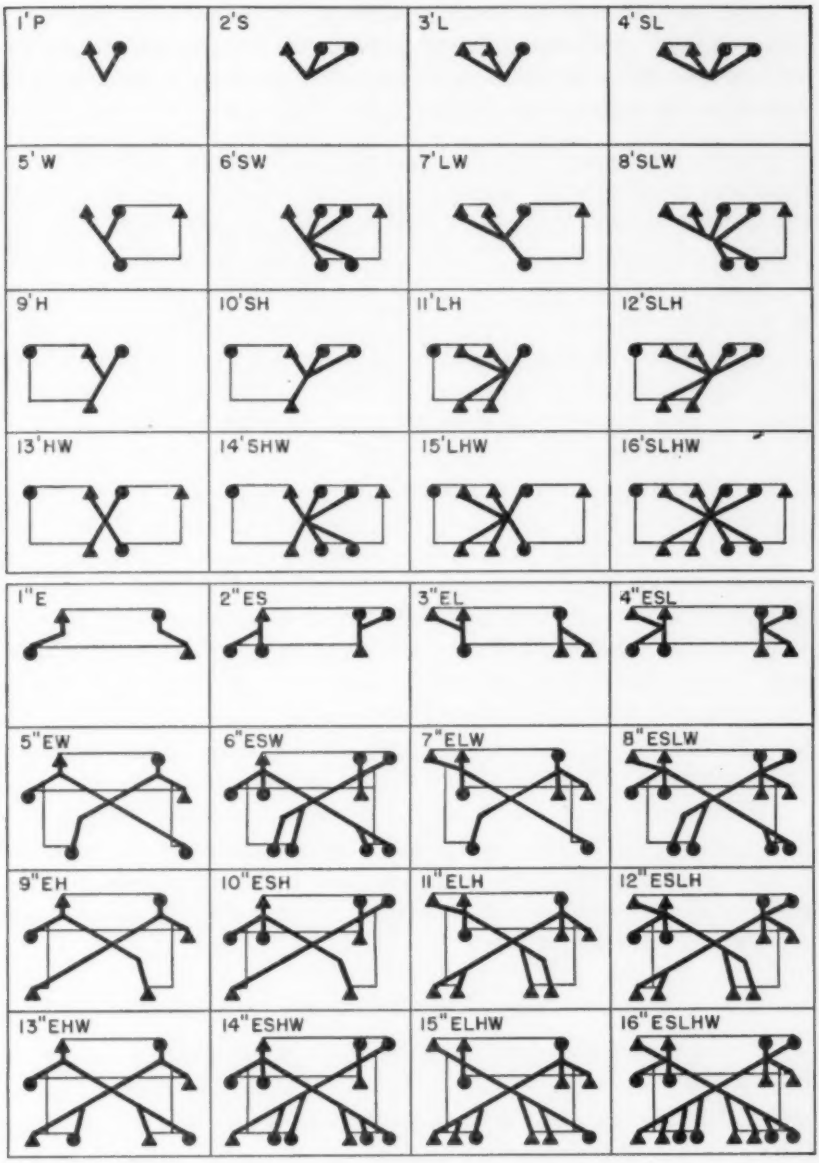


FIG. 1. PRIME AND DOUBLE PRIME SERIES.

Regarding the Primary as universally present we find that when we combine the other 5, one with another, we then have 26 Compounds. The Primary, the 5 Secondaries, and the 26 Compounds add up to a total of 32. These 32 forms of marriage either alone or in combination contain every known type of marriage.

Series. In figure 1 the diagrams for the 32 forms of sexual mating have been presented in a double series. The 16 which include Exchange are doubled forms of the other 16 which do not contain Exchange. Thus there are the Prime Series (1′–16′) and the Double Prime series (1″–16″).

Each of the two major series in turn contains 4 minor series, each of which is represented horizontally in a progression from left to right.

Each of the 32 diagrams of figure 1 shows the nexus of individuals (of the two families) who are involved in each sexual mating and therefore also represents a different nexus of genetic entities.

In a previous study (Aginsky, '35b, pp. 62-64) it was shown that in addition to the Primary and Secondary categories of sexual mating, there is also a tertiary category. It is a combination of plural Secondaries of certain kinds and results in such varieties of sexual mating as cross-cousins, grandparent-grandchild, and brother-sister. In certain cases the Primary disappears and the two or more Secondaries may continue through time without any new Primary occurring. The same is true in certain Compounds which result in Tertiaries. The Tertiary continues and the Secondaries or compounds of Secondaries disappear. The Tertiary then continues without the necessity of any Secondary being practiced.

When any type of sexual mating between related individuals (*e.g.*, grandparent-grandchild, cross-cousins, brother and sister) is considered, it must be borne in mind that there was previously a sexual mating, possibly also between related individuals, which brought each of these related individuals into being.

DERIVATION OF FORMULAE

It is apparent that each of the 32 diagrams consists of a nucleus of individuals who belong to two families. Each of the families is composed of individuals who are either male or female and who belong to the same or different generations.

For the present, *only* the sexual mating between the members of the two families (biological entities) will be considered. In a later section some examples of more than two families will be presented.

Thus we could state: in the Primary, there is present a sexual mating of one man of a family to one woman of another family, but no other sexual mating is present between members of these two families. The other individuals are sexually mated to members of different families.

In Sororate marriage we could say that there is present the sexual mating of one man of a family to the plural women of another family and that no other members of the two families are sexually mated to one another. We can phrase Sororate as the sexual mating of *a single Man* of *Family a* with *plural Women* of *Family b.*

However, unless mention is made of *Generation* one would not know whether one or more than one Generation is involved. To make the statement coincide with the diagram in this respect it is better to say: A *single Man* of *Family a* is sexually mated with *plural Women* of a *single Generation* of *Family b.*

However, this is an inclusive statement and does not exclude other sexual mating between or among other members of both families; therefore it is best to phrase the statement so that it is applicable in the other forms of sexual mating which are to be analysed as we proceed. Thus the total statement is:

A *single Man* of a *single Generation* (but *zero Woman*) of *Family a* is involved with *plural Women* of a *single Generation* (but *zero Man*) of *Family b.*

This can be abbreviated into a formula:

S = (MsGs) (WzGz) Fa ⩒ (WpGs) (MzGz) Fb

M	represents	Man
W	"	Woman (elsewhere W sometimes stands for mating with Wife's brother's daughter)
G	"	Generation
F	"	Family (biological entity)
a or b	"	particular families
z	"	zero or none
s	"	single or one
p	"	plural, or more than one
⩒	"	sexual mating (marriage) between or among

Upon analysis we find that every form of sexual mating contains the same factors. These factors we shall consider invariants. It has also

been found that these invariant factors are found in a universal interrelationship. That is, all of the factors are always present. The quantity of each factor may vary.

The universal equation. Each of the 32 diagrams represents the nexus of the male(s) and female(s) of the two biological entities who are directly involved in the particular variety of sexual mating. Each nexus results in what we shall term nucleus. Each diagram shows the number of individuals involved from each family. If we delete the quantitative differences of: s, p, and z, and the indications of Family, Fa and Fb, we then have as that which is common to all 32 formulae the following:

$$(MG)(WG) \vee (WG)(MG).$$

This formula indicates only that there is present a sexual mating relationship between and among the members of two biological entities without any indication of quantity. It is a universal formula.

Sex may be thought of as a force which is manifested in, for example, sexual intercourse with animals, with inanimate objects, with self, with members of the same sex, and with members of the opposite sex. It is this last category of manifestations—heterosexuality, upon which we have focused our investigation and analysis. Of all the manifestations in this category we are here concerned only with marital sexual relations which result in children, and thus "the family."

(We shall use "A" to designate the force Sex since "S" has been preempted for Sororate and "s" for single.) The universal equation is $A = (MG)(WG) \vee (WG)(MG)$.

If we give A some definite quantity we find that there is a corresponding quantity manifested in the right hand side of the equation, *e. g.*:

Universal equation	$A = (MG)\ (WG) \vee (WG)\ (MG)$
1′ Primary	$P = (MsGs)(WzGz)Fa \vee (WsGs)(MzGz)Fb$
2′ P plus Sororate	$S = (MsGs)(WzGz)Fa \vee (WpGs)(MzGz)Fb$
3′ P plus Levirate	$L = (MpGs)(WzGz)Fa \vee (WsGs)(MzGz)Fb$
4′ P plus S plus L	$SL = (MpGs)(WzGz)Fa \vee (WpGs)(MzGz)Fb$

We can tentatively conclude that each nucleus is the result of sexual mating and varies proportionately to the quantity of A.

Table 1 contains the 32 formulae. It is divided into the Prime and Double Primes series, each of which is subdivided into 4 minor series.

TABLE 1

Prime and Double Prime Series 32 Forms of Sexual Mating

1′	P	= (MsGs) (WzGz) Fa ⩒ (WsGs) (MzGz) Fb	
2′	PS	= (MsGs) (WzGz) Fa ⩒ (WpGs) (MzGz) Fb	Mono-generational
3′	P L	= (MpGs) (WzGz) Fa ⩒ (WsGs) (MzGz) Fb	males and females
4′	PSL	= (MpGs) (WzGz) Fa ⩒ (WpGs) (MzGz) Fb	
5′	P W	= (MsGs) (WzGz) Fa ⩒ (WsGp) (MzGz) Fb	
6′	PS W	= (MsGs) (WzGz) Fa ⩒ (WpGp) (MzGz) Fb	Mono-gen: males
7′	P LW	= (MpGs) (WzGz) Fa ⩒ (WsGp) (MzGz) Fb	Poly-gen: females
8′	PSLW	= (MpGs) (WzGz) Fa ⩒ (WpGp) (MzGz) Fb	
9′	P H	= (MsGp) (WzGz) Fa ⩒ (WsGs) (MzGz) Fb	
10′	PS H	= (MsGp) (WzGz) Fa ⩒ (WpGs) (MzGz) Fb	Poly-gen: males
11′	PL H	= (MpGp) (WzGz) Fa ⩒ (WsGs) (MzGz) Fb	Mono-gen: females
12′	PSLH	= (MpGp) (WzGz) Fa ⩒ (WpGs) (MzGz) Fb	
13′	P WH	= (MsGp) (WzGz) Fa ⩒ (WsGp) (MzGz) Fb	
14′	PSWH	= (MsGp) (WzGz) Fa ⩒ (WpGp) (MzGz) Fb	Poly-gen: males
15′	P LWH	= (MpGp) (WzGz) Fa ⩒ (WsGp) (MzGz) Fb	Poly-gen: females
16′	PSLWH	= (MpGp) (WzGz) Fa ⩒ (WpGp) (MzGz) Fb	
1″	P E	= (MsGs) (WsGs) Fa ⩒ (WsGs) (MsGs) Fb	
2″	PS E	= (MsGs) (WpGs) Fa ⩒ (WpGs) (MsGs) Fb	Mono-gen: males
3″	P L E	= (MpGs) (WsGs) Fa ⩒ (WsGs) (MpGs) Fb	Mono-gen: females
4″	PSL E	= (MpGs) (WpGs) Fa ⩒ (WpGs) (MpGs) Fb	
5″	P W E	= (MsGs) (WsGp) Fa ⩒ (WsGp) (MsGs) Fb	
6″	PS W E	= (MsGs) (WpGp) Fa ⩒ (WpGp) (MsGs) Fb	Mono-gen: males
7″	P LW E	= (MpGs) (WsGp) Fa ⩒ (WsGp) (MpGs) Fb	Poly-gen: females
8″	PSLW E	= (MpGs) (WpGp) Fa ⩒ (WpGp) (MpGs) Fb	
9″	P HE	= (MsGp) (WsGs) Fa ⩒ (WsGs) (MsGp) Fb	
10″	PS HE	= (MsGp) (WpGs) Fa ⩒ (WpGs) (MsGp) Fb	Poly-gen: males
11″	P L HE	= (MpGp) (WsGs) Fa ⩒ (WsGs) (MpGp) Fb	Mono-gen: females
12″	PSL HE	= (MpGp) (WpGs) Fa ⩒ (WpGs) (MpGp) Fb	
13″	P WHE	= (MsGp) (WsGp) Fa ⩒ (WsGp) (MsGp) Fb	
14″	PS WHE	= (MsGp) (WpGp) Fa ⩒ (WpGp) (MsGp) Fb	Poly-gen: males
15″	P LWHE	= (MpGp) (WsGp) Fa ⩒ (WsGp) (MpGp) Fb	Poly-gen: females
16″	PSLWHE	= (MpGp) (WpGp) Fa ⩒ (WpGp) (MpGp) Fb	

In brother-sister marriage (1MG1)Fa ⋁ (1WG1)Fa, the G1 represents the identical generation for both the brother and the sister. It is also possible to represent the marriage of any other two or more relatives.

In a later section we shall derive finite quantities and develop our formulae further. This will be done for both M-W and for G.

Substitution of Quantities for s *and* p. It is rather simple to substitute quantities for s and p, for example in a case where two men of Family a in the first generation are both married to one woman of

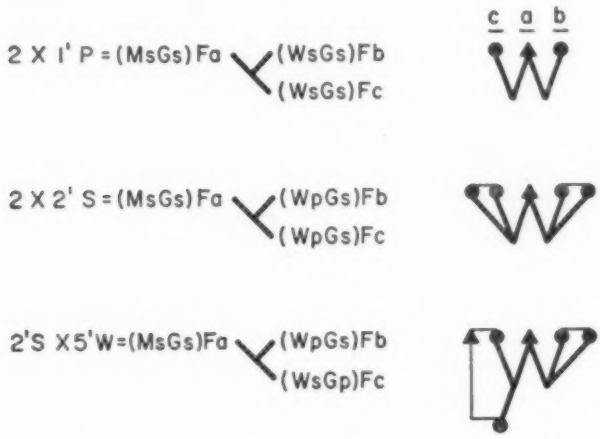


Fig. 2. Some Examples of Formulae Representing Sexual Mating of Individuals from More than Two Families.

one generation of Family b (fraternal polyandry) that nexus may be indicated as follows: (2MG1)Fa ⋁ (1WG1)Fb. The formula (4MG1)(2WG2)Fa ⋁ (2WG2)(1MG1)Fb, indicates that 4 men of the first generation of Family a are married to 2 women of the second generation of Family b and that 2 women of the second generation of Family a are married to one man of the first generation of Family b.

$$\begin{matrix}(2MG1)Fa \\ (1MG2)Fa\end{matrix}$$ mated with (4WG1)Fb or (2MG1)(1MG2)Fa ⋁ (4WG1)Fb

Both of these indicate the same nexus, namely, that 2 men of generation 1 and one man of generation 2 of Family a are married to 4 women of generation 1 of Family b. For further examples see figure 2.

PROGRESSIONS

Upon analysis of figure 1 we find that the progressions in the minor series are not quantitatively uniform. For example, 1′–4′ (P, S, L, SL), starts with Primary, goes to Sororate, then Levirate, and then Sororate + Levirate. By examination we find that Sororate (2′) and Levirate (3′) are opposite forms of sexual mating and appear to be of the same value quantitatively. Therefore, the progression should be from 1′ (Primary) to 2′ or 3′ (Sororate or Levirate) which should be on the

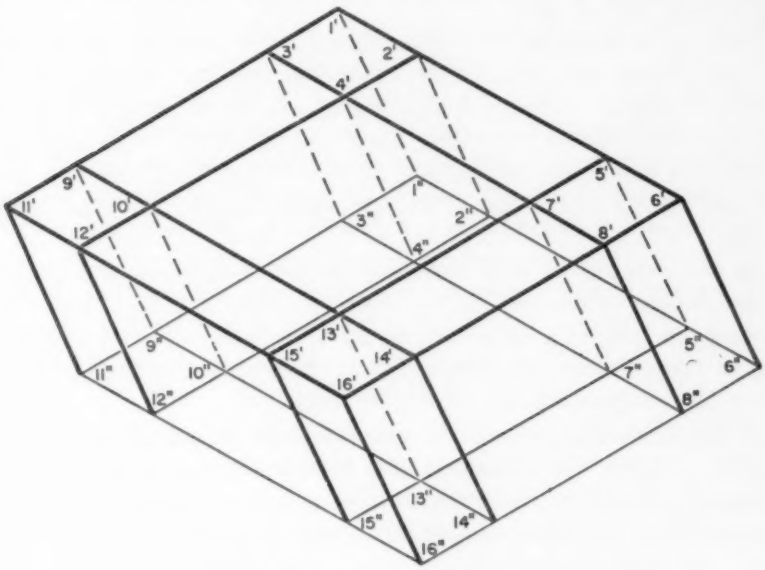


FIG. 3. PROGRESSIONS.

same level as each other and then to 4′, which is the total of 2′ and 3′. Thus the progression is: 1′ (2′–3′) and 4′, as represented in figure 3. Upon examination we find that the minor series 9–12 is the same as 1–4. Numbers 10 and 11 are of the same quantity and are opposites. The series 5–8 is opposite the series 9–12 but of the same value. Therefore the diagram should be arranged to indicate that these values are quantitatively parallel. The final minor series, 13–16, is greater in quantity than those which precede it but it is similar to them in that 14 and 15 are opposites.

Therefore, a table of progressions for each of the Prime and Double

Prime series could bring together the 4 progressions. Each level should be considered, tentatively, as containing those of the same quantity. The nuclear structures of the Double Prime series, are also presented in an identical 4-fold diamond pattern. It appears that in each of the 4-fold diamonds quantitative change would most easily follow along the diagonals. The line of change could easily branch at a right angle at any point and proceed along a new diagonal or it could reverse directions at any point. However, it would appear to require a greater amount of change for the direction to be at a 45° angle to these or to skip in other directions.

When we superimpose one diamond upon the other we can see that the change from one series to the other does not require too great an amount of change. The quantification of amount of change from one form of sexual mating to another along the diagonals, horizontals, and between the two major series, that is, the change from any one form of sexual mating to another of the 32, would require additional study, however.

As the quantity of A is increased or decreased there is a proportionate change in the nuclear structure of sexual mating. Therefore, a more exact table of progressions would have to be presented as a more complex three-dimensional form. The exact form of that "cube" would depend on the quantification of the 32 forms.

If the 32 contain all of the reported forms of sexual mating and in addition others which have not been reported as yet, it may very well be that we have succeeded in indicating a table of mathematical progressions which has some relation to actual changes in mating patterns.

CONSTANTS AND PROGRESSIONS

When we examine the diamond shaped chart (figure 3) and follow the diagonals, from top left to bottom right we find that for the Prime series:

In 1, 2, 5, 6, the quantity of the male factor MsGs is constant, while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 3, 4, 7, 8, the quantity of the male factor is MpGs and remains constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 9, 10, 13, 14, the quantity of the male factor is MsGp and remains

constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 11, 12, 15, 16, the quantity of the male factor is MpGp and remains constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

On the other hand, if we follow the diagonals in the diamond from top right to bottom left the female factor remains constant in each of the diagonals while the male factor increases progressively in each step.

We have here tables of progressions which indicate, not an infinite continuous series, but rather a finite mathematical progression based upon the quantities of the invariant factors of Gender (Man and Woman), and Generation. The quantities are s and p for both Man and Woman and Generation of each biological entity (family).

We now can see more easily 1) that the same factors and the interrelation of those factors are always present and 2) that a change in quantity of the force Sex (A) results in a corresponding change in the form of sexual mating and the resulting nuclear structure. It can also more easily be seen that the nuclear structures of sexual mating are directly proportional to the quantity of A.

PROBABILITY

Upon examination of the diagrams of figure 1, we find that the Primary is present in all 32 forms of sexual mating, while each of the 5 Secondaries occurs in every possible combination. Thus, each Secondary is found as an element in 16 of the forms, once by itself and 15 times in Compounds with one or more of the other Secondaries.

The forms in which different elements occur are shown in table 2. If each one of the 32 forms of sexual mating were found with equal frequency, this table of probabilities would show the frequencies of the elements. Excluding all other matters one would expect to find Sororate (or L, or W, or H, or E) in half of the matings, and in 15/32 of the cases it would be found as part of a Compound combined with other Secondaries. However, in order to achieve any worthwhile degree of correlation with actual cases, it would be essential to understand additional factors which are involved in determining the form of sexual mating present in any actual case.

An hypothesis. In populations which are evenly divided between males and females of sexual mating age it seems, although there may be

TABLE 2

Probabilities of Various Elements in the 32 Forms of Sexual Mating

Element	Total No.	Prime series																Double Prime series													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
P	32	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S	16		0		0		0		0		0		0		0		0		0		0		0		0		0		0		0		0
L	16			0	0			0	0			0	0			0	0			0	0			0	0			0	0			0	0
SL	8				0				0				0				0				0				0				0				0
W	16					0	0	0	0					0	0	0	0					0	0	0	0					0	0	0	0
S W	8						0		0						0		0						0		0						0		0
LW	8							0	0							0	0							0	0							0	0
SLW	4								0								0								0								0
H	16									0	0	0	0	0	0	0	0									0	0	0	0	0	0	0	0
S H	8										0		0		0		0										0		0				0
L H	8											0	0			0	0											0	0			0	0
SL H	4												0				0												0				0
WH	8													0	0	0	0													0	0	0	0
S WH	4														0		0														0		0
LWH	4															0	0															0	0
SLWH	2																0																0
E	16																	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
E S	8																		0		0		0		0		0		0		0		0
E L	8																			0	0			0	0			0	0			0	0
E SL	4																				0				0				0				0
E W	8																					0	0	0	0					0	0	0	0
E S W	4																						0		0						0		0
E LW	4																							0	0							0	0
E SLW	2																								0								0
E H	8																									0	0	0	0	0	0	0	0
E S H	4																										0		0				0
E L H	4																											0	0			0	0
E SL H	2																												0				0
E WH	4																													0	0	0	0
E S WH	2																														0		0
E LWH	2																															0	0
E SLWH	1																																0

a variety of forms of sexual mating in practice, that the forms would be in balance. The relationships between forms indicated in the diamond of figure 3 may be of aid in this matter.

One might expect to find, for example,

2′ and 3′ being practiced simultaneously or both together as 4′, or
5′ and 9′ " " " " " " " 13′, or
6′ and 11′ " " " " " " " 16′, or
all 16 " " " " all " " 16′, and
similarly for the Double Prime series.

However, in populations where the ratio of males and females is not equal one might expect to find different forms practiced. With a preponderance of males, L and/or H might be found with no other form, or in combination with other forms depending upon the ages, sex ratio, size of the population etc.

THE RESULTANTS AND QUANTIFICATION

The previous diagrams represent only the nuclei of the 32 varieties of sexual mating. We shall now present the resultants, *i. e.*, the kinship

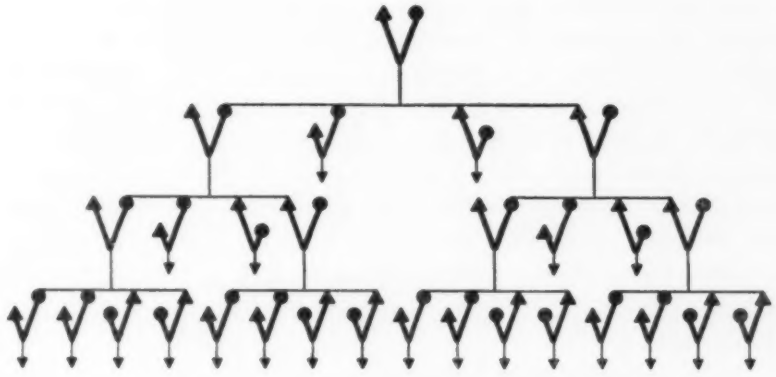


FIG. 4. 1′P. GENETIC AND CULTURAL.

systems, of the Primary and the 5 Secondaries, and also 4′ $(2' + 3')$ as an example of a Compound.

The Primary diagram is presented first (figure 4) and represents both the genetic and the cultural. It is a genetic chart with 4 children indicated for each married female. Since the number of children is proportional to the number of female parents, 4 children will be shown for each female parent in the diagrams which follow.

Figures 5, 7, 9, 12 and 14 are presented as the "cultural" result of each form of sexual mating and marriage wherein those children who have at least one parent in common are placed in the same sibling group. However, an example of the genetic results of each sexual mating form is illustrated by separate diagrams (figures 6, 8, 10, 13 and 15), wherein the genetic lines for each married individual are indicated and the sibling relationships of each sibling group are indicated by breaks in the horizontal line.

By comparing the kinship systems with one another, one may note the differences in the nuclear family structures, constellations of relatives, number of relationships, number of individuals in each relationship, and resultant genetic relationships.

The kinship systems which are presented diagramatically should be considered as "pure" laboratory constructs. Actual kinship systems may be compared with them. Attention is directed to the repetitive pattern within each diagram. Due to the limitation of space, arrows have been used to indicate repetitive descending groups.

One may calculate the quantity in any generation by multiplying the number in one group of a descending line by the number of descending lines. For example, in 1′, there are 4 descending lines. When we count the number of new families involved—marriage groups, number of descendants, number of strangers, etc.—in one descending line in the second generation we find: 4 families, 4 descendants, 4 strangers or a total of 8. When we multiply these figures by 4 descending lines we then have 16, 16, 16, and 32.

Table 3 presents the numbers for each of 1′, 2′, 3′, and 4′. A comparison indicates that there is a difference of genetic "spread" or "contraction."

Before going on to 5′ and 9′ where plural generations are involved we shall discuss the quantitative aspect involved in the numbers of men, women and generations.

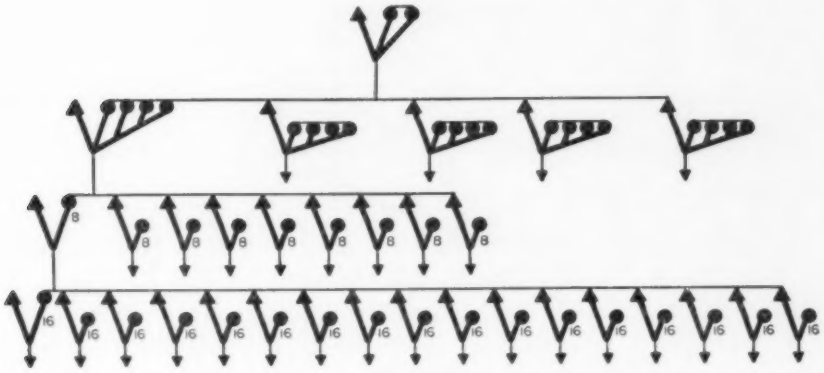


FIG. 5. 2′S. CULTURAL.

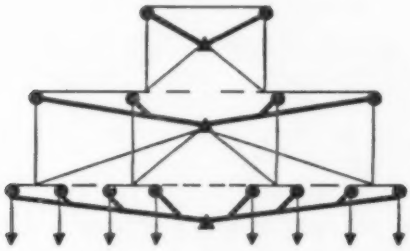


FIG. 6. 2′S. GENETIC.

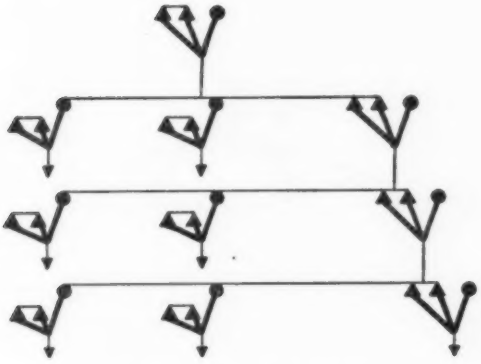


FIG. 7. 3′L. CULTURAL.

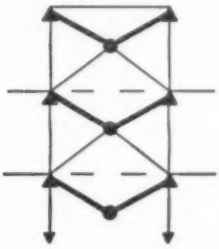


FIG. 8. 3'L. GENETIC.

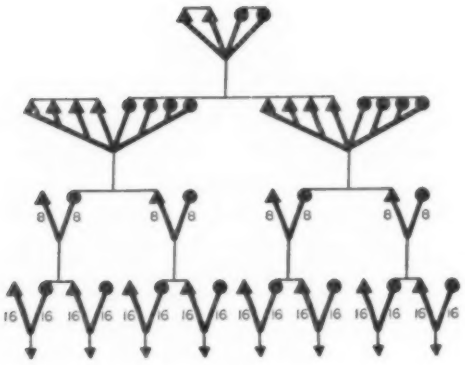


FIG. 9. 4'SL. CULTURAL.

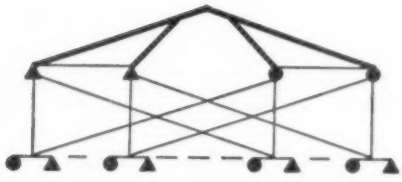


FIG. 10. 4'SL. GENETIC.

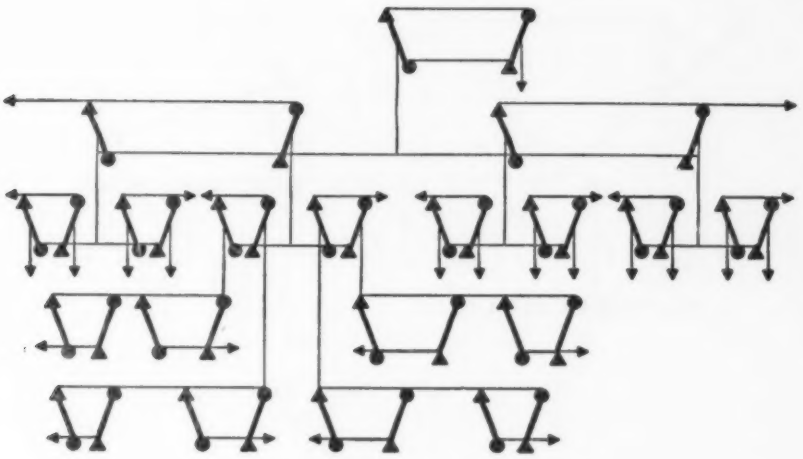


FIG. 11. 1″E. GENETIC AND CULTURAL.

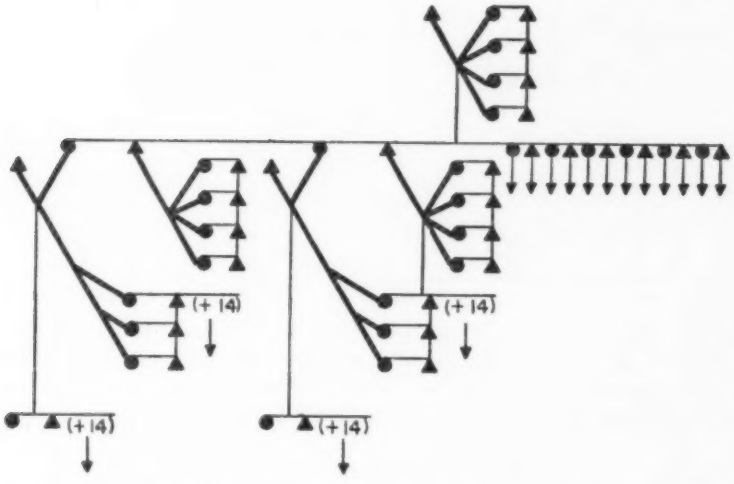


FIG. 12. 5′W. CULTURAL. Since a man may impregnate for 60 years he can mate with women of 4 generations. Note that, to a woman, her own children and her brother's daughter's children have the same father (her own husband); to a man, his sister's children and his daughter's children have the same father.

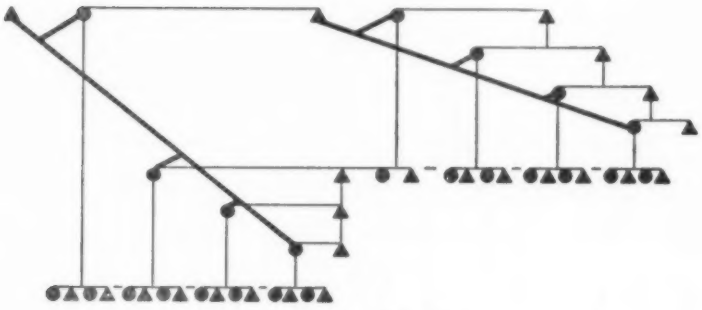


FIG. 13. 5'W. GENETIC.

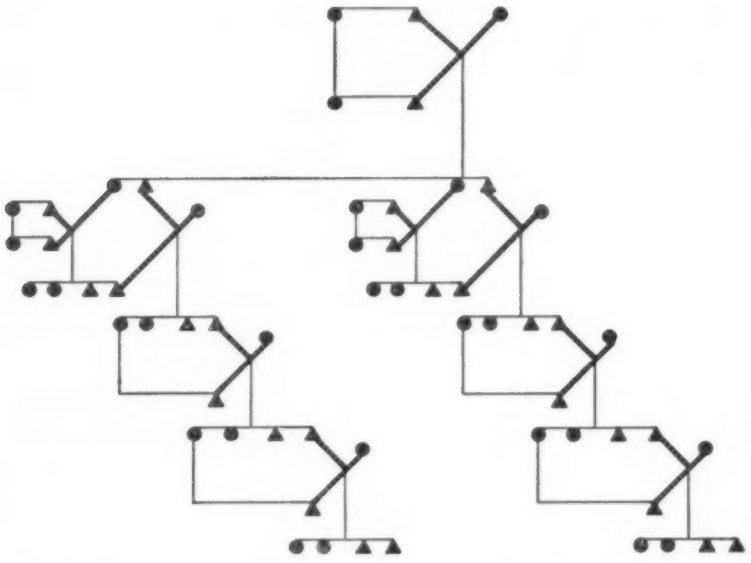


FIG. 14. 9'H. CULTURAL. Since a woman can bear for 30 years she can mate with men of two generations. Note that, to a man, his own children and his sister's grandchildren have the same mother (his own wife); to a woman her brother's children and her son's children have the same mother.

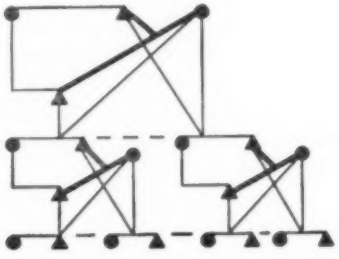


FIG. 15. 9'H. GENETIC.

TABLE 3

Quantitative Comparison 1', 2', 3', and 4'

	1'				*2'*				*3'*				*4'*			
G	F	D	ST	T	F	D	ST	T	F	D	ST	T	F	D	ST	T
1	4	4	4	8	5	8	17	25	3	4	5	9	2	8	8	16
2	16	16	16	32	45	80	325	405	9	12	15	27	4	32	32	64
3	64	64	64	128	765	1,440	11,565	13,005	27	36	45	81	8	128	128	256
TT	84	84	84	168	815	1,528	11,907	13,435	39	52	65	117	16	168	168	336

G Generation
F new families involved and sexual mating groups
D number of descendants
ST number of strangers involved
T total strangers and descendants
TT total of all in third generation

SUBSTITUTION OF QUANTITIES FOR THE SYMBOLS M, W, G, AND THE VALUES s AND p

The quantities below have been chosen arbitrarily for easy computation and to indicate a method by which quantification may be accomplished in the formulae:

Initial child-bearing by women 15 years of age; initial impregnation by men 15 years of age. (Therefore each generation amounts to 15 years.) Terminal child-bearing by women 44 years of age. (Thus a woman bears children for 30 years.) Terminal impregnation by men 74 years of age. (Thus a man impregnates for 60 years.) Therefore the value of WG is 2 (Generations) and the value of MG is 4 (Generations) when plural generations are involved.

Upon this basis in 5′ W the man may marry the W in 4 generations of his wife's family, while in 9′H the woman may marry the M in two generations of her husband's family. When W and H are found in the same compound (13′, 14′, 15′, 16′), the result is that a perpetual system comes about. We shall indicate this as 2+ for men and 5+ for women.

It can be seen that where Gs is involved, *e.g.*, MsGs ⋁ WsGs (and this condition obtains for the lifetime of the man and the woman) the result is determined by the factor W which limits the factor M, and arbitrarily using 4 children for each woman there will be but 4 children. In 2′ S, MsGs ⋁ WpGs, there will be 8 children and again the result is limited by the factor W.

However, where Gp is the quantity the matter is more complicated as can be seen in table 4. Here quantities have been substituted for the symbols in each of the formulae in the Prime series. Across the top of the table are listed both s and p for W, M, and G.

Since there are different combinations of parents in each of the 16 forms of sexual mating in the Prime series (and also in the Double Prime series) the genetic composition of the descendants will also be different in each of the 16. Although the total of Qf and Qm, that is parents, may be the same there is a different genetic resultant. For example in 3′L there are 2 fathers who are brothers as compared with 9′H where there are 2 fathers who are mother's brother and sister's son.

Should we apply the quantities presented in this section to cultural genetics we find that at the end of 180 years, counting generations through the first born of the first born upon the basis of 15 years per generation there would be a total of 12 generations preceding the last

individual. If the count is through the last born female of the last born female and using 30 years as the gauge there would be but 6 generations, and if the count were through the last born male of the last born male with the gauge being 60 years there would be but 3

TABLE 4

The Substitution of Quantities for Symbols in the Prime series

	Q of f				Tf	Q of m				Tm	Qch	T in Nu F	Tfm
	Ms	Mp	Gs	Gp		Ws	Wp	Gs	Gp				
1′ P	1		1		1	1		1		1	4	6	2
2′ S	1		1		1		2	1		2	8	11	3
3′ L		2	1		2	1		1		1	4	7	3
4′ SL		2	1		2		2	1		2	8	12	4
5′ W	1		1		1	1			4	4	16	21	5
6′ SW	1		1		1		2		4	8	32	41	9
7′ LW		2	1		2	1			4	4	16	22	6
8′ SLW		2	1		2		2		4	8	32	42	10
9′ H	1			2	2	1		1		1	4	7	3
10′ SH	1			2	2		2	1		2	8	12	4
11′ LH		2		2	4	1		1		1	4	9	5
12′ SLH		2		2	4		2	1		2	8	14	6
13′ HW	1			2+	2+	1			5+	5+	20+	27+	7+
14′ SHW	1			2+	2+		2		5+	10+	40+	52+	12+
15′ LHW		2		2+	4+	1			5+	5+	20+	29+	9+
16′ SLHW		2		2+	4+		2		5+	10+	40+	54+	14+

Q of f equals the number of fathers
Tf " total fathers
Q of m " the number of mothers
Tm " total mothers
Q of ch " quantity of children
T in Nu F means Total in Nuclear Family and is the addition of Tf, Tm, and Qch. Tfm is the total of Tf and Tm and also represents the quantity of genetic entities.

generations. At the end of 180 years, although all 3 individuals (first born of first born, last born female of last born female, and last born male of last born male) would be born in the same year, each would have an entirely different quantitative generational genetic history. In short, the genetic effects of males and females in the population would vary considerably.

By now it is clear that the progressions illustrated in figure 3 must be redrawn on the basis of 4 generations for males and 2 generations for females, (figs. 12-15).

THE WIDER FOCUS

As we construct larger nuclear sexual mating groups, we are simultaneously enlarging the sexual mating groups for all the members of the two sibling groups. Thus, the size and composition of each spouse's

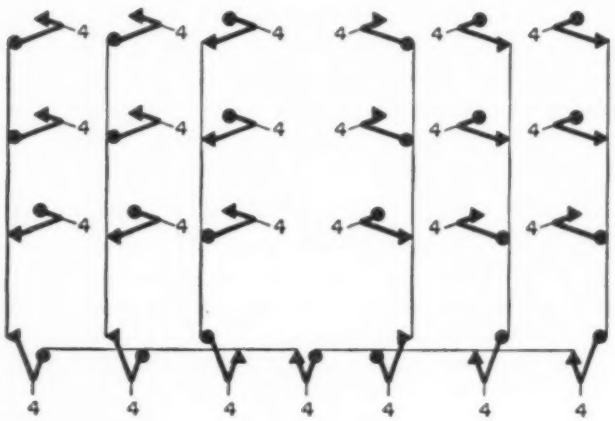


FIG. 16. MODEL USING 1'P. There are 25 nuclear families present, each consisting of 6 members. There are 25 fathers, 25 mothers and 100 children totalling 150 persons.

sibling's affinal family varies in each of the 7 cases, the variation is proportional to the form of sexual mating, and is correlated with the size and composition of the nuclear family. Figures 16-20 illustrate this point.

For the sake of simplicity we shall present the diagramatic results only for 1′, 2′, 3′, 4′ and 1″. The numeral in the box indicates the number of children of the sexual mating group.

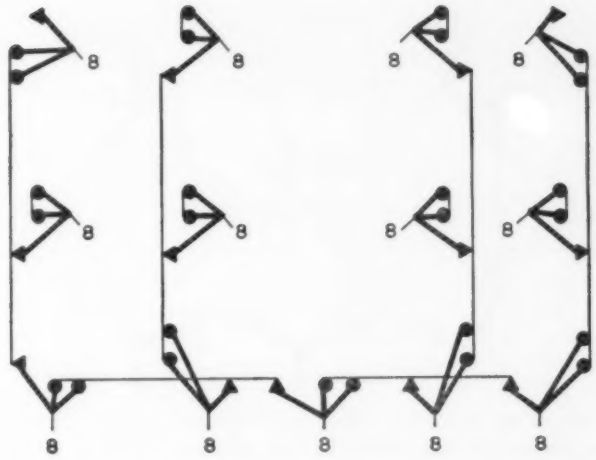


FIG. 17. MODEL USING 2'S. There are 13 nuclear families present, each consisting of 11 members. There are 13 fathers, 26 mothers and 104 children totalling 143 persons.

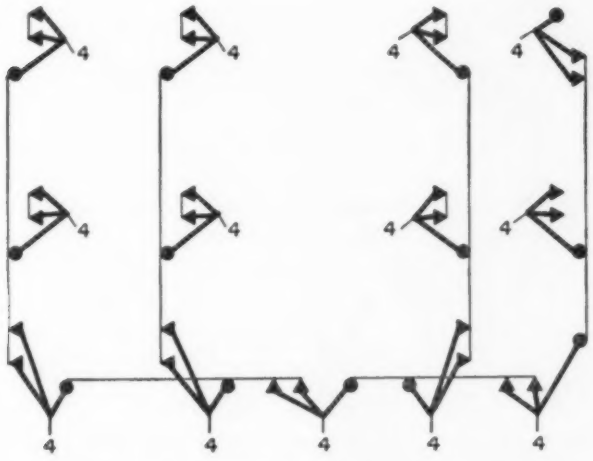


FIG. 18. MODEL USING 3'L. There are 13 nuclear families present, each consisting of 7 members. There are 26 fathers, 13 mothers and 52 children totalling 91 persons.

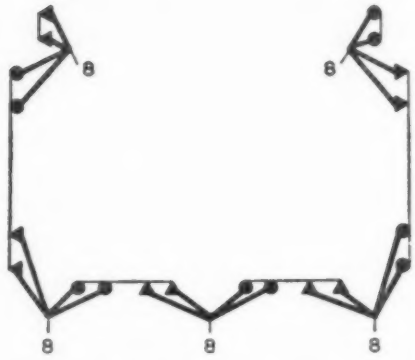


FIG. 19. MODEL USING 4'SL. There are 5 nuclear families present, each consisting of 12 members. There are 10 fathers, 10 mothers and 40 children totalling 60 persons.

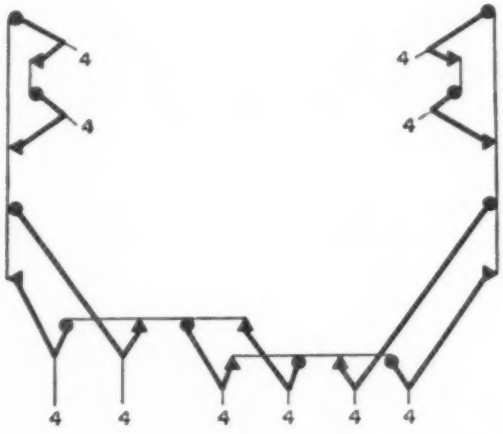


FIG. 20. MODEL USING 1"E. There are 10 nuclear families present, each consisting of 6 members (actually 5 double families of 12 each). There are 10 fathers, 10 mothers and 40 children totalling 60 persons.

Within the present focus the differentials of factors M and W even out to some extent and, as diagramed, it appears that the greater the quantity of the force Sex (A) the smaller the total number of individuals involved.

TABLE 5

Comparative Table

	1′	*2′*	*3′*	*4′*	*1″*
1. Number of individuals in each sexual mating group	2	3	3	4	2
2. Number of children in each nuclear family	4	8	4	8	4
3. (no. 1 + no. 2) total individuals in nuclear family	6	11	7	12	6
4. Number of sexual mating groups	25	13	13	5	10
5. (no. 1 × no. 4) total individuals-sexual mating groups	50	39	39	20	20
6. (no. 2 × no. 4) total children	100	104	52	40	40
7. (no. 5 + no. 6) total individuals	150	143	91	60	60

The greater the amount of A, the larger the particular sexual mating group, the fewer the number of separate sexual mating groups and nuclear families, the smaller the total quantity of individuals involved, and the smaller the population.

Therefore the smaller the total population the larger the sexual mating group would tend to be. In short, one would anticipate a correlation between the total size of the population and the form of sexual mating.

RELATIONSHIPS, AFFINAL AND CONSANGUINEAL, COMPOSITION AND QUANTITY

In the Primary 1′, the maximum number of relationships remain in the affinal families since only one individual has been taken from each of the two families to construct the new nuclear family, *i. e.*, husband and wife. To the man, and also to the woman, of this sexual mating group, each affinal family consists of the remaining individuals of various relationships. For the sake of simplicity we shall include only

the members of the nuclear families. Thus we shall utilize only the members of the sexual mating groups and their children.

Table 6 contains the relationships which are present or absent and the quantity of individuals in each relationship for 1′, 2′, 3′, 4′ and 1″. Both the man (column II) and the woman (column III) relate to the identical individuals. Thus there are always at least two relationships present in regard to each individual, one for the man and one for the woman except for husband and wife. (Column I is the total of II and III.)

Column I contains the quantities of relationships for 1′. The nuclear family of 1′ contains 6 relationships. Each succeeding line contains 8 relationships. At the bottom of the column we find the total 6/32. The 6 represents the number of relationships in the nuclear family while the 32 represents the total relationships in the combined affinal and consanguineal families.

When we follow across the grand total line we find, under IV, the quantities of relationships is repeated under 1′ and then the grand totals for 2′, 3′, 4′ and 1″. The line above entitled "Individuals" contains the total of A and the grand totals of BCDE. Thus under IV 1′ is found 6/36. The 6 represents the number of individuals in the nuclear family while the 36 represents the total number of individuals in the combined affinal and consanguineal families.

In 2′, the man, being married to his wife's sister, does not have a female affinal nuclear family and therefore does not have some of the relationships present for the man as in 1′, namely, wife's sister, wife's sister's husband, wife's sister's son and daughter. The woman does not have a female consanguineal nuclear family. Here her sister is her co-wife and her sister's children are her husband's children and are members of her own nuclear family. However the man now has:

4	wife's brother's wives	instead of 2
8	wife's brother's sons	instead of 4
8	wife's brother's daughters	instead of 4

The woman now has:

4	brother's wives	instead of 2
8	brother's sons	instead of 4
8	brother's daughters	instead of 4

Table 6 contains the relationships which are present or absent and

the quantity of individuals in each relationship for 1′, 2′, 3′, 4′ and 1″. Both the man and the woman relate to the identical individual. Thus there are always at least two relationships present in regard to each individual, one for the man and one for the woman.

It is to be noted that all of the relationships are present in 1′ and that all of the relationships for the nuclear family (line A) are present in all of the cases. However, the relationships for B are lacking in 2′, for D in 3′, and for both B and D in 4′.

When we look at F (grand total) for relationships, we find that 1′ and 1″ have the same quantity 6/32 but are different in C and E due to the double relationships in 1″. 2′ and 3′ have the same quantity of relationships 6/24 but are different in B and D. Thus none of the five contain the same constellation of relationships. When we carry this through all 32 forms of sexual mating and the resulting kinship systems we can then see that each of the 32 is a different kinship system.

When we compare the 5 cases in regard to the quantity of individuals we find that 1′ contains a greater quantity of individuals than relationships because there are 2 or 4 individuals in certain of the relationships. 2′ contains the largest quantity of individuals despite the fact that it contains fewer relationships than 1′. The constellation of relatives varies from one type of sexual mating to another, certain relationships are sometimes absent while others are consolidated.

From table 6 it appears that the greater the quantity of the force Sex (A) the fewer the relationships but the greater the quantity of individuals in some of the relationships.

As the quantity of A is increased there is a proportional decrease in the number of relationships. It appears that it is important to differentiate between the relationship and the quantity of individuals in the relationship.

SUMMARY

From the reported forms of sexual mating 6 forms have been presented as being basic: the Primary which is composed of one man and one woman of different families and 5 Secondaries: Sororate, Levirate, Wife's brother's daughter, Husband's sister's son, and Exchange. Combinations of two or more Secondaries result in a total of 26 possible Compounds making a total of 32 forms in all. These can be divided into a Prime series lacking Exchange matings and a Double Prime series involving Exchange. Within each series the forms may be represented

TABLE 6

Comparisons of Quantities of Relationships and Individuals

		I	II	III	IV				
		Q of Relationships	Man to his	Woman to her	Q Ind. per Relationship				
					1'	*2'*	*3'*	*4'*	*1''*
Nuclear Family	A	6	w		1	2	1	2	1
				h	1	1	2	2	1
			s	s	2	4	2	4	2
			d	d	2	4	2	4	2
			Total individuals		6	11	7	12	6
Affinal and Consanguineal Families	B	8	w si	si	1	0	1	0	1
			w si h	si h	1	0	2	0	1
			w si s	si s	2	0	2	0	2
			w si d	si d	2	0	2	0	2
			Total individuals		6	0	7	0	6
	C	8	w b	b	2	2	2	2	1–1[a]
			w b w	b w	2	4	1	2	1–1[b]
			w b s	b s	4	8	2	4	2–2[c]
			w b d	b d	4	8	2	4	2–2[d]
			Total individuals		12	22	7	12	6–6
	D	8	b	h b	1	1	0	0	1
			b w	h b w	1	2	0	0	1
			b s	h b s	2	4	0	0	2
			b d	h b d	2	4	0	0	2
			Total individuals		6	11	0	0	6
	E	8	si	h si	2	2	2	2	1–1[b]
			si h	h si h	2	1	4	2	1–1[a]
			si s	h si s	4	4	4	4	2–2[c]
			si d	h si d	4	4	4	4	2–2[d]
			Total individuals		12	11	14	12	6–0*
Grand Totals	F			Individuals	6/36	11/44	7/28	12/24	6/24–6
		6/32		Relationships	6/32	6/24	6/24	6/16	6/32

* Double Relationships.

		To the Man		To the Woman
1''	[a]	1—w b and si h si	—is the same individual—	b and h si h
	[b]	1—w b w and si	—" " " "	—b w and h si
	[c]	2—w b s and si s	—" " " "	—b s and h si s
	[d]	2—w b d and si d	—" " " "	—b d and h si d

in a diamond such that on one axis a progression from matings with a single female of one generation to multiple females of one generation to a single female from each of multiple generations to multiple females of multiple generations would be shown. On the other axis the corresponding progression for males would be shown and the whole 16-fold diamond could be superimposed on the corresponding one for the Double Prime series. Hypothetically one might expect changes in customary mating practices to follow the progressions indicated by such a model.

The 32 varieties of sexual matings were further analysed and seen to consist of invariant factors with quantitative variation, male(s) and female(s) of generation(s) of one family mated to those of another family. These can be expressed as a universal formula in which they are equated with a quantity (A). In general any change in A would be accompanied by a change in the form of sexual mating. Furthermore, a change in A would correlate positively with changes in the number of individuals in the sexual mating group, nuclear families, individuals in the nuclear family, individuals in the affinal and consanguineal nuclear families, individuals in the total population, relationships, individuals in the relationships, and families in the total population.

LITERATURE CITED

AGINSKY, BURT W. 1935A The mechanics of kinship. American Anthropologist *37*: 450-457.

——— 1935B Kinship systems and the forms of marriage. American Anthropologist, Memoir *45*.

——— 1939 Time levels in societal analysis. American Anthropologist, *41*: 416-432.

——— 1940 Interacting factors in the Maori family. American Anthropologist, *42*: 195-210.

BOOK REVIEWS

Genetica Medica. I^um Symposium Internationale, 6-7 Septembris MCMLIII. Edited by LUIGI GEDDA. Pages xxiii + 467, Istituto Gregorio Mendel, Rome, 1954. Price not indicated.

This book is a double first. It is the first volume of a new series, *Analecta Genetica.* And it is the proceedings of the First International Symposium of Medical Genetics.

The work opens with the remarks of Pope Pius XII, translations of which are given in Italian, English and German. Perhaps the sentence of the Papal remarks most significant to the geneticist *as* scientist is: "L'appréciation des questions purement spécifiques est laissée à la compétence de votre science" (p. xviii).

The proceedings of the symposium are divided into 12 *Relazioni* and 18 *Comunicazioni.* Two of the papers are in French (F), 5 in English (E), 7 in German (G) and 16 in Italian (I). Surnames of the authors, shortened titles, and an abbreviation of the language of the *Relazioni* are, in the order printed: Gedda, Medical genetics, I; Gianferrari, Longevity, I; Kallmann, Health and mental disorder, E; Franceschetti & Klein, Identification of heterozygotes, F; Lehmann, Endocrine diseases, G; Torrioli & Romei, Pernicious anemia, I; Waardenburg, Papilla leporina, vitiligo, and Leber's optic atrophy, E; von Verschuer, Longevity, G; Lamy & Bienenfeld, Discondrosteosy, F; Hanhart, Geography of human genes, G; Grebe, Pathology of the skeletal system, G; and Mollison, Hemolytic disease of the newborn, E.

The *Comunicazioni* are: Andreani, Conti & Monicelli, Germinal aplasia and hypogonadism, I; Arrigoni, Cresseri & Lovati, Polycystic disease of the kidney, I; Canziani & Riccobono, Tests for musical talent, I; Ceppellini, Aminoaciduria, I; Casa, Diabetes, I; De Biase, Dental anomaly discordant in MZ twins, I; Dell'Acqua, Somatic, biochemical, and physiological observations on MZ twins, I; Gatto, Drepanocytosis in Italy, I; Hoffer, Maxillo-facial anomaly, I; Jucci, Baldness, I; Korkhaus, Teeth and facial skeleton, G; Lombardi, Cardiovascular disease, I; Maltarello, Cooley's anemia in MZ twins, I; Oostenrijk, Poliomyelitis acuta anterior, E; Pavoni, Morganti & Avanza, Blood groups of Pesaro, I; Moench & Schaeuble, Laurence-Moon-Bardet-Biedl syndrome in twins, G; Stecher, Herberden's nodes, gout, and ankylosing spondylitis, E; and Testa, Histological study of twins, G.

The papers of this symposium vary greatly in purpose and scope. All are of interest and substance to medical genetics. My vote for the one paper of this group which might be of most general interest to the human biologist would go to E. Hanhart, Beiträge zu einer humangenetischen Geographie (pp. 165-187). Aside from the genes of the red blood cell antigens, previously little had been written in summary form on the population distribution of human genes.

J. N. SPUHLER

University of Michigan

Clinical Versus Statistical Prediction: a Theoretical Analysis and a Review of the Evidence. By P. E. MEEHL. Pages vii + 149, University of Minnesota Press, Minneapolis, 1954. $3.00.

In this slim, attractively produced book Dr. Meehl has set out to examine a burning problem in clinical psychology. The first part is devoted to a statement of the problem, to the examination of logical issues, to the reactions of other writers, *e.g.*, Sarbin, and to an analysis of clinical intuition. The later chapters are concerned with empirical evidence on the relative effectiveness of the two methods and with problems of quantification of clinical observations.

At the commencement Dr. Meehl points out an important distinction between the *discriminative* and the *structural* use of statistics. The former is represented by tests of significance. The latter is concerned with more complex psychological constructs, used to 'explain' behavior. As Dr. Meehl correctly emphasizes, "the prototype of this use of statistics is factor analysis." The term "actuarial," which Dr. Meehl uses in his chapter headings as opposed to "clinical," is intended to include this more potent use of statistics and not merely the blind, non-psychological, non-theoretical use of actuarial statistics.

The definition of "clinical," however, is not reached so early or clearly. The reader finds himself asking: "Do we mean conclusions reached without conscious awareness of the logical or mathematical processes?" "Or without awareness of all the observations being taken into the judgment?" "Or by use of facts and principles precisely similar to that of the psychometrician but involving *mental* calculation instead of formal computation?" "Or by use of mathematical relationships not yet employed by statistical psychologists?" At times Dr. Meehl seems to be toying with this last meaning, which would be a quite

unwarranted use, for *any* conceivable functional relationships of observations and predicted criteria can be equally employed, implicitly by the clinician and explicitly by the personality statistician. It would be ridiculous to restrict the latter, for example, to first order equations or to non-pattern considerations. Dr. Meehl's proposition that the statistician may also use "prediction from a class" indicates that he rejects this restriction. But reference, for example, to the Pattern Similarity Coefficient, r_p, applied to patterns of functionally unitary factors, would have illustrated a realm apparently little known to clinicians (as the writer later states), and would have left no remaining doubt of the writer's theoretical position.

At times Dr. Meehl flirts with a still more untenable definition of the clinical method, when he proceeds in his argument to represent the clinical method by a mature, full-fledged psychotherapist and the actuarial method by a young woman clerical worker substituting figures in given equations! (The impression is somehow given that she barely graduated from high school and is rather weakminded.) If we are to compare the two methods, let us at least have trained psychologists in both positions, each fully aware of whatever psychologically predictive principles can be applied to the observations! A similar sop to the clinician is gratuitously given by Dr. Meehl when he quotes Dr. Reik's account of a remarkable and (as events proved) true inference drawn by Dr. Reik from a very small symptom, and asks what the statistician would do in such a case. I'm afraid the statistician would first ask Dr. Reik to try to remember all the unrecorded instances where an inspired guess proved *wrong*! To the reviewer the only satisfactory definition of the clinical method is that it proceeds in part upon unconsciously perceived data and in part by inexplicit or unconscious reasoning, *i.e.*, "intuitive" application of logical and mathematical relations. In so doing, it uses wholistic and pattern formulations which, prior to the development of factor analysis and pattern calculation, were not used by the psychometrist, and are admittedly *still* not used by most psychometrists "trained" for clinical work.

On reaching the empirical treatment Dr. Meehl musters no fewer than nineteen studies—all very interesting reading—in the great majority of which he ultimately concludes that the statistical predictions have been significantly better than the clinical ones. Few of these studies have been even tolerably well designed to answer all that could be answered.

Little attempt has been made to see that the clinical and the statistical representatives have reached equal maturity of training in their studies, or even that they are supplied with the same data or testing time of the subjects, or that the area of possible prediction is well sampled; but Dr. Meehl shrewdly appraises these uncontrolled variables. Actually, as he points out, in these unequal competitions it is always the clinician who has been supplied with more time and data and who yet fails to reach the same efficiency of prediction. Many insufficiently known findings come up in this evaluative study, *e.g.*, Baron's finding that in the Rorschach experiment "neither a mechanical use of the usually mentioned signs, nor a subjective sorting by four Rorschach experts (with access to the protocols) showed any correlation with therapeutic outcome."

The final chapters on quantification with use of patterns, etc., are cut very short, just when the statistician would be interested in resolving alternative possible conceptualizations of the significant predictions from patterns made by Dr. Meehl and others. In any case, due to the instability of mere individual "items" in tests the psychologist is going to be more interested in patterns based on psychologically meaningful temperamental and dynamic *factors* than in patterns in arbitrary *items*. With so much work still needing to be done on primary personality factors and the trying out of simple linear predictions from these psychological entities, it is intellectually very easy but strategically premature to be insisting that additive relations are not enough. As Meehl well says, "There is a lot of loose talk around these days about non-linear relations. I do not doubt that there are a large number of such in the behavior domain, but we ought not to browbeat the statisticians with this phrase until we know more about where these non-linear dependencies occur and how much they pay off predictively over and above the much-maligned linear regression system."

A careful theoretical and empirical evaluation of this problem has been long overdue. It is hard to imagine anyone better qualified by combination of clinical and statistical viewpoints than Dr. Meehl to tackle this involved question. Certainly clinicians are likely to give more serious attention to these unfavorable findings when they come from a person of established reputation in their own realm than from a clinically uninterested statistician. Actually, in the reviewer's opinion, Dr. Meehl has maintained the balance very fairly, but perhaps with a little more charity to his kin than others! The sooner this painful self-knowledge

is accepted by the hard pressed clinician, the sooner will true remedies, in terms of better research-based tests and more insightful prognostic procedures, become possible.

RAYMOND B. CATTELL

University of Illinois

Human Heredity. By JAMES V. NEEL AND WILLIAM J. SCHULL. Pages 361, University of Chicago Press, Chicago, 1954. $6.00.

This is a general textbook of human heredity with emphasis on methodology. We think it is outstanding and certain to play a large role in the training of future human geneticists. It is intended for readers who wish to learn not only accumulated facts and theories, but research procedures as well. Much of the book is devoted to various ingenious devices of Weinberg, Fisher, and others for handling difficult problems in Mendelian ratios, linkage, etc. that would be simple in animals where experimental matings are possible. The principal tool is the maximum likelihood method, which is introduced early and used consistently throughout the book. We think this is a good idea, for although it is debatable whether this is always the best method, there is the real merit of giving the student one asymptotically efficient procedure that is applicable to a wide variety of problems.

The authors start out on an optimistic note by pointing out some of the advantages of man as an object of genetic study, rather than following the usual practice of deploring his obvious disadvantages. Then comes a very short chapter on cytology. The nature-nurture problem is introduced early and discussed throughout the book with no attempt to hide its complexity. There is a clear and balanced presentation of the methods and results of mutation rate study without undue emphasis on the authors' own minority view that average rates are high. After several chapters on formal genetics the book ends with sections on epidemiology, counseling, forensic genetics, and eugenics.

The chapter on physiological genetics is good and we should like to see it expanded in future editions. The section on blood groups is surprisingly brief, but perhaps this isn't too serious because of the excellent books available on this subject (*e. g.,* Race and Sanger). The section on testing Mendelian ratios with various types of ascertainment bias is the clearest account we have seen. But we think there is a serious omission; no method is provided for the case where ascertainment is

incomplete but known, and since the simply applied Weinberg proband method is appropriate and of fairly high efficiency we should like to have had it included. The chapter on linkage gives only the Penrose sib pair method. Here is one of the few places where we believe the authors could have been more critical by pointing out the liability to error and the low efficiency of this method. Unfortunately, methods of detecting and measuring linkage are complex, but perhaps the gift the authors have shown for clear writing could be used to present some better methods. The authors regard as the main purpose of linkage study its possible use in prognosis. We should prefer to emphasize linkage relations as a research tool for distinguishing between phenotypically similar, but genetically different entities or for calling attention to such heterogeneity. The chapter on twin studies is particularly good in pointing out pitfalls in interpretation for the uncritical. Our only complaint is that, in using Holzinger's H as a measure of heritability, they fail to note that this is quite different from heritability as used by animal and plant breeders. Finally we like the chapters on counseling and eugenics, perhaps because we share the authors' reservations.

Although the book is very modern in its approach, it is already out of date in some areas. This is, of course, a statement about the rate of advance in knowledge rather than a criticism of the authors, who, by being responsible for some of the advances, have shortened the useful life of their book. Some new works that are not included are: the relation of thalassemia and sicklemia to malaria resistance, evidence for mutation at the MN locus, new work on hemoglobins, possible position effects and the f antigen in the Rh series, new evidence for selection in the ABO blood groups, and new factors governing prothrombin formation. We are already looking forward to a second edition.

In subject-matter this volume is similar to Stern's *Principles of Human Genetics*. It differs in being more concise, in having a greater emphasis on research methods, and in assuming more knowledge of genetics, statistics, and differential calculus on the part of the reader. The authors make no apology for this. They say: "The complexities of the study of human heredity are such that knowledge of certain branches of mathematics is no less essential to the serious student of human heredity than to the astronomer or physical chemist. The text which attempts to disguise this fact is, in the long run, doing the student a disservice." Here, again, we agree emphatically.

JAMES CROW AND NEWTON MORTON

University of Wisconsin

Atlas of Men. A Guide for Somatotyping the Adult Male at All Ages. By W. H. SHELDON, with the collaboration of C. WESLEY DUPERTUIS AND EUGENE MCDERMOTT. Pages xvi + 357, Harper, New York, 1954. $10.00.

When the mariners of the days of Vasco da Gama and Columbus left their native shores and trusted to the wind of discovery they brought back with them a variety of hard-earned treasures. There was gold perhaps, and strange animals, and sometimes overcredulous tales; but for those who came after them the most important thing they brought was an atlas, a map of countries other than their own. And so perhaps with all exploration; each of us has to sail on his own private voyage from his own private island to meet others inhabiting other islands which may seem strange or engulfing or hostile. For this more personal journey, we need an atlas too, because if we know something of the natures of people different from ourselves and expect of them behaviour also different we shall be much less likely ourselves to be frightened or hostile, righteous or overbearing. Just as the scientific study of primitive peoples has had the effect of increasing our understanding and sympathy with them and has enabled us to learn more about ourselves, so the scientific study of the differences in constitutional make-up of people in a single culture can lead to increased understanding and sympathy between people of different temperament, physique and aptitude. In this, Sheldon's *Atlas of Men* has perhaps the usefulness, partial and imperfect, that the first primitive maps of the ocean had for the early seafarers.

The book is primarly an atlas of photographs, and as such it must be judged. It consists of 1175 sets of somatotype photographs, beginning with the 117 and progressing through the ones in endomorphy by the steps 126, 127, 136, 145, 154, 162, 163, 171 and 172. Then come the two's in endomorphy arranged in a similar order; then the three's, and so on. The material on which the atlas is based is quite staggering. During the last 20 years Sheldon and his collaborators have photographed and somatotyped some 46,000 men, of all ages (from 18 to 65). On the first 4000 the procedure of somatotyping and the scales of the three components were standarized: this formed the basis for the *Varieties of Human Physique,* published in 1940. Now, in the more extensive material, 88 somatotypes have been found, or 505 somatotypes if 13-point rating scales are used for each component instead of the original 7-point scales. Each of these 505 physiques is presented in the atlas, and nearly

all of them at two or three different ages—typically about 20, 40 and 60 years. The somatotype of each of the five regions of the body is given for each picture, so that the dysplasia of each is also recorded. In addition a list is given of pictures which together form scales for androgyny of physique and for texture-component.

The pictures themselves measure about $3\frac{1}{2}'' \times 4''$, that is, about two-thirds of the size at which they were originally taken. Some loss of detail is brought about by this reduction, but much more by the halftone process itself: one of the minor frustrations of all workers in this field is to see their most cherished photographs, which in the original showed every faint muscle shadow and every individual hair, reduced on publication to a uniform shapeless grey (with X-rays the situation is still worse). It is idle to pretend that these reproductions replace for the actual somatotyper a key file of real photographs, though the publishers have probably done as well with them as is possible at present. They are certainly a considerable improvement over those in the *Varieties of Human Physique*. Inevitably in such a huge collection the quality of the originals varies considerably, both as to photographic and posing technique. On the first page, for example, No. 2 shows a lot of detail that in No. 4 has been quite obliterated, and the pose of No. 762, compared with that of No. 772, leaves a good deal to be desired.

These, however, are the technical criticisms of the fellowcraftsman. Let it be said at once that this atlas is a magnificent achievement, a feat of sustained and meticulous endeavour which has no parallel whatever in the previous history of the subject. Even to the casual glance the range of variation in human morphology displayed cannot fail to be awe-inspiring. Turn, for example, to page 71 and compare the 172 and the 216. Or contemplate Nos. 369 and 1078 (a 271 and a $6\frac{1}{2}$ $2\frac{1}{2}$ $1\frac{1}{2}$), who are almost identical in height and weight but totally dissimilar in shape and in tissue composition. Look at No. 1038, and then No. 138. "If sometimes you find yourself doubtful about the ultimate fate of man" writes Sheldon "and in need of reassurance as to the vastness of his adaptability, try to contemplate the enormous range of adaptive potentiality that must inhere in such radically different somatotypes as the 651 and the 145. Then recall that these two represent only two points in a far-flung, continuous extension in three dimensions. Perhaps we do well to carry the great range of somatotype variation that this present culture appears to afford. Therein may lie the best insurance against an untimely ending of the human play."

With the pictures goes a page of text for each somatotype. This includes a chart of the particular somatotype's position on the conventional somatotype distribution diagram, a graph of the average height/$\sqrt[3]{\text{weight}}$ at each age from 18 to 65, and a table of weight-for-height at each age. These graphs and tables are invaluable, and with them we have for the first time standards of weight for height which are not nonsensical. They may be too detailed for present clinical use, but certainly from them broader categorisations with appropriate standards could quite easily be made. They are admittedly based, as is all the atlas and the somatotyping, on cross-sectional material, and Sheldon is rightly insistent that this makes them subject to revision when longitudinal data to replace them become available. Clearly we need pictures of the *same* 354 at 20 and 40 and 60 and not pictures of persons aged 40 and 60 who, in the somatotypers opinion, probably at 20 were the same somatotype as the 354 20-year old. Those involved in the field should now already be planning another atlas on longitudinal lines, which will need yet more determination, and yet more sympathetic farsightedness from those in control of funds and facilities. They will also be planning to take X-rays as well as photographs, skinfold thicknesses as well as weight and height, and a whole variety of physiological and psychological measurements. But that is another step forward, and here we are concerned with the nature and imprint of the first step only.

Below the tables of weight and height for each somatatype is half a page of general comment. It seems to me a pity that this does not include the detailed physical description of the somatatype given in the *Varieties of Human Physique,* which instead has to be used in conjunction with the atlas if the best use is to be made of the pictures. The general comments however, as one would anticipate in any book of Sheldon's are always interesting and lively, though he specifically warns the reader that they are to be taken as "impressions provocative of new research . . . and not confused with established fact." They reveal in particular an interest in longevity and in the applications of somatotyping to clinical medicine and psychiatry.

One minor feature of the book will doubtless come in for a disproportionate amount of comment. Sheldon has given an animal totem to each somatotype, an amusing echo of Aristotle and Della Porta. For each somatotype there are a few lines in italics ostensibly describing the animal, but endeavouring to refer obliquely to some aspect of the somatotype about to be described. Sheldon calls these italicised lines

"self-evidently an accompaniment in a minor key to be read or omitted accordingly to the reader's taste." The metaphor is apt; not all listeners are able to distinguish the major from the minor mode, and those who cannot had much better leave the italics alone. They should perhaps bear in mind, however, that a disinclination to smile does not of itself signify the taking of a subject *au sérieux,* and vice versa. Inevitably, some of the totems are far-fetched and largely beside the mark, but in others the short and telling phrase—"killed by a careless gesture, but for all that they can sting resolutely"—reveals the sharp pen of the born novelist.

Preceding the atlas proper there are some thirty pages of introduction. In them are discussed, with equanimity and a wit quite innocent of the rancourous overtones which marred some pages of an earlier book, the history of somatotyping, the effect of age and nutrition on somatotype, the scales for use in somatotyping women, and the relative frequency of each somatotype. One remark here, however, is quite misleading. Sheldon implies (pp. 5, 14) that the three somatotype components are orthogonal. This is by no means the case; they are negatively interrelated to the extent that (judging from the figures of the earlier book) only between one-half and one-third of the variance of each component is left when the other two components are held constant. It is a pity that joint frequency distributions of the components are not given in the atlas; the frequency distributions for single components are there and show that mesomorphy and ectomorphy are very nearly normally distributed, while endomorphy has a long tail of high values and a shortage of low ones (that is, positive skew).

This obliquity of the components is one of the arguments against the usefulness of somatotypes as a method of classification. It is not a very strong argument, for it may really be that the somatotype components are more than statistical abstractions and have roots in physiological and genetical mechanisms. Though biometrically it is somewhat harder to handle oblique than orthogonal factors, the ultimate choice between them depends on the biological and not the statistical background. Only further experiment and more knowledge can decide the ultimate fate of Sheldon's classification; data alone will do so, not debate.

Meanwhile "by taking a careful weight history and using the height-weight criteria included in this volume, by matching your standardised photographs against these plates, and by studying the published checklists, it will be found possible to somatotype." Of one

thing Sheldon may remain certain: this atlas is a landmark in the study of human physique and behaviour that will not be submerged by the tides of many decades. Somatotyping has changed the study of physique immeasurably and brought an order and precision that the field never knew before, but which now it will keep whatever change the future may bring. Somatotyping has also been a major influence in bringing about the present renewed interest in human constitution and human biology amongst medical men and biologists. These are great achievements for Sheldon and Dupertuis and their later colleagues to have accomplished, and they have put us all most surely in their debt.

J. M. TANNER

Sherrington School of Physiology,
St. Thomas's Hospital, London

Perspectives in Physiology: An international symposium. Edited by ILZA VEITH. Pages 171, American Physiological Society, Washington, D. C., 1954. $3.00.

At the XIXth International Physiological Congress, held in Montreal in 1953, sixteen leading representatives of physiological science were brought together to exchange thoughts on the future and limitations of physiology and to present a picture of the past and present state of physiology in their respective countries. The essays in this little book are the presentations made in the introductory sessions of the symposium.

The book is divided into two sections, the first on "Limitations and Future," the second on "The Present Scene." The tone of the former is philosophical, of the latter documentary. It is a varied fare. Some of the essayists are preoccupied with the problems of communication and organization posed by the rapid expansion and fragmentation of physiological science. A few broach the more difficult question of *quo animo.* Thus Homer Smith of the United States exhorts us to again become *physiologoi* in the original Greek sense, concerned with a knowledge of nature in any of her aspects. And Alexander von Muralt of Switzerland says, in regard to the question: what is life?, that although we have dispensed with the old vitalism we are still faced with an ultimate restriction and must exclaim with Du Bois-Reymond "ignoramus, ignorabimus." For K. M. Bykov of Russia the lines along which physiological science should be developed are those defined by I. P. Pavlov. To Bykov it was Pavlov who introduced the "synthetic, materialistic"

approach in contrast to the "analytic, idealistic" approach of Western Europe.

In the section describing the present status of physiology in various countries the essays range from catalogs of personnel and laboratories to dispassionate analyses of the toll taken by war and political upheaval.

MORTON I. GROSSMAN

V. A. Hospital,
Los Angeles, Calif.

The Biologic Effects of Tobacco. Edited by ERNEST L. WYNDER. Pages xiii + 215, Little, Brown and Company, Boston, 1955. $4.50.

Smoking is a typically complex problem of human biology. Of its many aspects the present volume covers the chemical and pharmacological fundamentals, and the medical implications (allergies, cardiovascular system, gastrointestinal tract, and neoplastic diseases). The chapter devoted to the "cause and effect" in epidemiological research should help to clarify the important concept of causation in the etiology of disease. E. C. Hammond defines a cause of a disease as a totality of conditions that is sufficient to produce it. Because of the ever-present individual variability in reactions to a given set of environmental conditions, a statistical interpretation of causation is favored and a causative factor is defined as "any condition which, in a given situation, increases the probability that a specific event will occur."

The compilation of the volume was stimulated by the current debate on the theme "To smoke or not to smoke." The 8 authors present a critical review of the available evidence, together with detailed references to original investigations. The work will serve well to bring the medical and scientific professions up to date on the subject.

While on some points our present knowledge is fairly definitive, on other points additional research is called for. Acute effects in normal individuals, long-term role in the etiology of degenerative diseases, and effects in patients with specific diseases must be clearly differentiated. The incidence of diseases suggesting association with smoking varies tremendously. Except for a few, very rare diseases, the specific reactions (including reduced peripheral blood flow) are not involved in the etiology of abnormalities of the cardiovascular system, even though the use of tobacco influences unfavorably the clinical course of established diseases,

e. g., of hypertension. On the other hand, lung cancer, in the etiology of which tobacco smoking (especially cigarette smoking) is held to play a definite role, is among the most common causes of cancer deaths in males (p. 110).

To the human biologist, tobacco consumption is of interest as one of the facets of the mode of life that exhibits large differences between individuals, between the sexes, and between geographic areas. To some, smoking is a highly treasured means of relaxation, while others regard it as an annoyance or a "sin." In any comprehensive account, the positive value attached to tobacco consumption can not be disregarded. There are on record verified observations that prisoners on the verge of starvation traded their meager nourishment for a few puffs. Some patients, suffering from diseases of the peripheral blood vessels, warned repeatedly against using tobacco, will continue to smoke in spite of the fact that smoking brings on agonizing pain from gangrenous ulcers and necessitates multiple amputations.

A cigarette may serve as a protest against parental control, a cigar as an outward symbol of social status. Smoking, as a facet of the overall mode of life, has rich and inadequately explored implications for cultural anthropology, the study of personality development, and, in its abnormal aspects, for psychiatry. How well substantiated is the impression that heavy smokers are tense, "nervous" individuals? How effective a relief of "nervousness" does smoking actually provide? Is nicotine the only factor in the enjoyment of smoking? As an "oral phenomenon," how is smoking related to appetite and weight control? Observations are available indicating that cessation of smoking among excessive smokers, who were underweight and complained of anorexia, leads to a gain of weight. However, there are no adequate data for samples of "normal" individuals.

More generally, we may ask: In what biologically relevant ways do different categories of smokers differ from non-smokers? To this question the present volume gives answers limited largely to considerations of disease. Scientific clarification of such problems as the relationship of tobacco consumption to personality, body weight and body composition must await further research. However, further work is needed on such basic problems as the carcenogenicity of tobacco smoke, the carcenogen being so far unidentified, and on the very nature of the association between smoking and lung cancer (*cf.* J. Berkson, Proc. Staff Mayo Clinic, *30:* 319-348, 1955).

JOSEF BROŽEK

University of Minnesota

BOOK NOTICES

FUNDAMENTALS OF RADIOBIOLOGY. By Z. M. Bacq and P. Alexander. xii + 389 pp. Academic Press, Inc., New York, 1955. $6.50. In the age of nuclear fission, the use of radiation in the treatment of cancer and the problem of avoidance of serious exposure hazards to health and life itself provide a strong practical stimulus to research in this field, requiring the pooling of the resources and methods of the physicist, chemist, biologist and clinician. The volume aims at an overall survey of the present, widely scattered information. Beginning with the physics of ionizing radiations, the survey ends with observations on human beings. Genetical phenomena, biochemistry of irradiated living organisms, and the action of the endocrines (pituitary, adrenal glands) in radiation sickness are also considered. Close to 1000 papers are quoted.

HISTORY OF THE PRIMATES. By W. E. Le Gros Clark. 119 pp. British Museum, London, 1954. 4 Shillings. This is a fourth edition of a readable and authoritative introduction to the study of the fossil man.

THE LUNG. By Julius H. Comroe, Robert E. Forster, Arthur B. Dubois, William A. Briscoe and Elizabeth Carlsen. viii + 219 pp. The Year Book Publishers, Inc., Chicago, 1955. $5.50. A book on clinical physiology and pulmonary function tests, written for physicians and medical students. Its aim is to explain in simple words and diagrams those aspects of pulmonary physiology that are important to clinical medicine.

THE HUMAN ORGANISM. By Russell Myles De Coursey. vii + 550 pp. McGraw-Hill Book Company, Inc., New York, 1955. $5.75. An elementary introduction to human anatomy and physiology, richly illustrated, with a glossary, suggested readings (principally books), and questions appended to each chapter.

RECEPTORS AND SENSORY PERCEPTION. By Ragnar Granit. xi + 369 pp. Yale University Press, New Haven, Connecticut, 1955. $5.00. These Silliman lectures, presented by one of the most prominent workers in the field, are a reflective "comment on the achievements of a quarter of a century of work by electronic methods on the special senses." The author had a significant share in the experimental investigations concerned with senses as interpreters of the external world. At the same time, the action-current analysis of the function of sensory organs was treated as a part of general neurophysiology. Of particular interest is the last chapter on sensory discrimination and integration. With its 50-odd pages of references, this volume is the best available summary of a fascinating but highly specialized field.

LABORATORY INSTRUMENTATION IN PSYCHOLOGY. By William W. Grings. vi + 282 pp. The National Press, Palo Alto, California, 1954. $4.75. Intended as introduction to the subject, at a moderately advanced level. Has three

general chapters (recording systems, timing and counting, biolectrical phenomena), three chapters on special senses (audition, vision, other senses), and chapters on human learning and perception. Gives references to more specialized and advanced sources. A more comprehensive treatment of the subject-matter in the future editions would be useful.

PSYCHOMETRIC METHODS. By J. P. Guilford. ix + 597 pp. McGraw-Hill Book Co., Inc., New York, 1954. $8.50. The pioneering first edition (1936), addressed to graduate students of psychology, combined the specific problems of psychological measurement (psychophysics, psychological scaling methods, mental tests) and the more general statistical treatment of quantitative data. With the statistical methods presented in *Fundamental Statistics in Psychology and Education* (1950), the second edition of *Psychometric Methods* is focussed on measurement in psychology, its theory, procedures, and the more specific statistical operations (such as the derivation of a factor matrix from a correlation matrix by factor-analysis methods).

VITAMINS IN THEORY AND PRACTICE. By Leslie J. Harris. xxii + 366 pp. Cambridge University Press, London, 1955. $6.50. This book, first published in 1935, grew out of popular lectures given at the Royal Institute, London, in 1934. The aim of the fourth edition remains to present, in a readable form, an up-to-date story of vitamin research.

GENETICS AND HUMAN HEREDITY. By J. Ben Hill and Helen D. Hill. xiv + 526 pp. McGraw-Hill Book Co., Inc., New York, 1955. $6.50. A text designed for students in elementary genetics. Examples and illustrations are drawn about equally from the plant and animal kingdoms.

HUMAN PHYSIOLOGY. By Bernardo A. Houssay, Juan T. Lewis, Oscar Orías, Eduardo Braun-Menéndez, Enrique Hug, Virgilio G. Foglia, and Luis F. Leloir. xvi + 1177 pp. McGraw-Hill Book Co., Inc., New York, 1955. $12.00. The appearance of the second edition, within 4 years, of this comprehensive textbook indicates that it has found a firm place in a highly competitive market. Written for medical students, it will serve well as a reference book for physicians and for scientists interested in facts and fundamental principles of modern physiology. The authors are teachers as well as productive investigators. The fact that they did not have to depend for describing the major portion of the *globus physiologicus* solely on literature (English, as well as French, Italian, German and Spanish) but could draw on first-hand experience, constitutes one reason for the appeal of this book.

STATISTICS AND MATHEMATICS IN BIOLOGY. Edited by Oscar Kempthorne, Theodore A. Bancroft, John W. Gowen, and Jay L. Lush. ix + 632 pp. The Iowa State College Press, Ames, Iowa, 1954. $6.75. The present volume grew out of the need for synthesizing concepts and methods of biology with those of mathematics (development of quantitative theories) and statistics (economy of description, including condensation of observations, prediction). The 44 papers were presented at a biostatistics

conference, held at Iowa State College in 1952. They fall into 5 groups: general biometric principles and procedures, changes in population numbers, estimation of population size, evaluation of biological responses, and genetical analysis of populations.

THE JOSIAH MACY, JR. FOUNDATION 1930-1955: A REVIEW OF ACTIVITIES. xi + 174 pp. Josiah Macy, Jr. Foundation, New York, 1955. A review of activities designed to "initiate, stimulate, develop and support scientific investigations of the fundamental aspects of health, of sickness, and of methods for the relief of suffering." The Foundation has played a significant role in the advancement of knowledge in medical and social sciences. Most important, perhaps, it provided liberal support for interdisciplinary collaboration essential for the integration of knowledge obtained in the separate sectors of the study of man.

ELECTROCHEMISTRY IN BIOLOGY AND MEDICINE. Edited by Theodore Shedlovsky. xii + 369 pp. John Wiley and Sons, Inc., New York. Chapman and Hall, Ltd., London, 1955. $10.50. Living organisms are able to transform chemical eneregy into electrical signals. The study of these signals provides important fundamental information about the normal function of nerves and muscles, and aids the physician in the diagnosis of the disordered function of heart and brain. The volume grew out of the 1953 symposium sponsored by the Electrochemical Society. The 18 papers were contributed by physicists, chemists, biologists, and clinical investigators. They present, in a highly technical fashion, the results of electrochemical researches on membranes, cells, ionic activity, polarography of plasma proteins, electrocardiography and electroencephalography.

BIOMATHEMATICS: THE PRINCIPLES OF MATHEMATICS FOR STUDENTS OF BIOLOGICAL SCIENCE. By Cedric A. B. Smith, xv + 712 pp. Hafner Publishing Co., New York, 1954. $13.50. This is the third edition of W. M. Feldman's *vademecum* on the application of mathematics to biology. Numbers, shapes and chances are all considered in the quantitative description of biological phenomena, even though here the emphasis is on arithmetic and geometry rather than on statistical methods. From a brush-up on elementary rules and tricks of arithmetic, the reader is led all the way to the mathematics of the rates of change, various ways of solving equations and the study of matrices.

ESSENTIALS OF BIOLOGICAL AND MEDICAL PHYSICS. By Ralph W. Stacy, David T. Williams, Ralph E. Worden and Rex O. McMorris. xiv + 586 pp. McGraw-Hill Book Co., Inc., New York, 1955. $8.50. A well organized textbook in a rapidly growing field, intended for use in courses on biophysics offered to students of biology (as one of the foundation blocks of their science), physics (as a facet of applied physics), and medicine (as a basis for some forms of therapy). It contains the following sections: Fundamental concepts, Mechanical systems (bones, muscles; effects of high accelerations on human organism), Heat, Sound, Light, Gas physics, Flow of fluids, Electrical phenomena, Nuclear physics, and Theoretical biophysics. Six appendixes.

GENERAL ENDOCRINOLOGY. By C. Donnell Turner. xi + 553 pp. W. B. Saunders Co., Philadelphia, 1955. Endocrinology is presented as a basic biological science, with internal secretion regarded as a mechanism operating throughout the whole range of plant and animal life and physiologically important during the developmental period as well as in adult individuals. With reference to the separate glands of vertebrates, anatomical features, biochemistry of the particular hormone, and the physiological effects are presented in turn. Photographs illustrating typical endocrine disturbances in man and animals are provided, but the clinical aspects of endocrinology are not treated extensively. In comparison with the first edition (1948), the principal addition is a chapter on adaptive reactions to stress.

THE MAMMALIAN FETUS: PHYSIOLOGICAL ASPECTS OF DEVELOPMENT. Cold Spring Harbor Symposia on Quantitative Biology, Vol. 19, xii + 226 pp. Biological Laboratory, Cold Spring Harbor, New York. $8.00. The primary aim of the conference was to evaluate the present knowledge about the morphological, physiological and biochemical factors affecting the mammalian, especially the human, embryo. Emphasis was placed on mechanisms involved in the somatic and visceral development essential for normal living before and just after birth, even though pathology (such as radiation effects) was also considered. Twenty-seven papers by American and European authors, with synthetic chapters (biochemical and physiological evaluation) and a review of implications for the management of the newborn infant.

THE MICMAC INDIANS OF EASTERN CANADA. By Wilson D. Wallis and Ruth Sawtell Wallis. xv + 515 pp. University of Minnesota Press, Minneapolis, Minnesota, 1955. $7.50. The field work among the Micmac was carried out in 1911, 1912, 1950 and 1953. The purpose of the early work was to obtain a detailed ethnographic account. The recent studies were focussed on cultural changes.

PHYSIOLOGICAL ACOUSTICS. By Ernest Glen Wever and Merle Lawrence. xii + 454 pp. Princeton University Press, Princeton, New Jersey, 1954. $10.00. Hearing involves a series of events—mechanical, electrophysiological (electrochemical), and psychological (perception of sound). The volume is a highly competent presentation of the initial, mechanical segment of this series, focussed on the function of the middle ear as an acoustical transformer and on sound conduction in the cochlea. Clinical applications (conductive deafness, otosclerosis) are considered and the present status of auditory theory is reviewed. Eighteen pages of references.

JOSEF BROŽEK

University of Minnesota

INDEX TO VOLUME 27

(Asterisked entries are titles, some slightly abbreviated.)

Age differences in—incidence of dental caries, 179; life expectancy, 204 ff., susceptibility to growth retardation, 260 ff., taste sensitivity, 157; thoracic form, 65 ff.
*Aginsky, B. W., Theory of Cultural Genetics, 301-29
Ainu, 186, 257
Albritton, E. C., 143
Anastasi, A., 243-6
Army and Air Force officers, longevity among retired, 125-37
Artificial limbs, 149-50
Australasians, blood groups among, 253 ff.

Banfield, W. G., 231-2
Basal metabolism and—body composition, 22 ff.; oxygen consumption, 25-7
Birth—effect on height and weight of month of, 138 ff.; weight in wartime, 268 ff.
Blood—groups, distribution of, 236-7, 253-7; absence of sickle-cell gene in Melanesia, 284-93
Body—composition and basal metabolism, 22 ff.; measurements of school children, 239-41
Bone, physiology of, 233-4
Bones, sexual diagnosis of isolated, 12 ff., *see also* Fossil bones
*Brožek, J.—book notices, 250-2, 343-6; book reviews, 143, 341-2; and F. Grande, Body Composition and Basal Metabolism, 22-31

Caries, dental—distribution of, 211 ff.; incidence in Micronesia, 179; susceptibility of children to, 1 ff.
Cattell, R. B., 331-4
Chicago, life expectancy in, 202 ff.
Children—body measurements of, 239-41; retardation in growth of, 258 ff.; susceptibility to dental caries of, 1 ff.
Clark, W. E. Le Gros, 241-3
Connective tissue, 231-2
Constitution, 336-40
Coronary heart disease, 144-5
Crow, J., and N. Morton, 334-5
Culture and genetics, 301-29

Depopulation of a Micronesian island, 161 ff.
Disease—and depopulation, 176 ff.; follow-up study of, 90 ff., 121 ff., 294 ff.

Equipment design, 248-9
Estes, W. K., *et al.*, 50-1

Europe—blood groups in, 253-ff.; population increase in the racial stock of, 187-9; retardation in growth of children of, in wartime, 258 ff.
Ewing, J. F., 235-6

Fat—accumulation, 53 ff.; and weight gain, 39 ff.; patterning, 75 ff.
Fatigue, a study of, 247-8
Fingernails, growth of, 274-83
*Fitt, A. B., Height and Weight according to Month of Birth, 138-42
Floyd, W. F., and A. T. Welford, 247-8, 248-9
Fossil bones—in evidence of human evolution, 241-3; from Natchez, Mississippi, 193 ff.

Gantt, W. H., 50-1
*Garn, S. M., 237-9—Relative Fat Patterning, 75-89; and R. V. Harper, Fat Accumulation and Weight Gain in the Adult Male, 39-49
Gedda, L., 330-1
Genetics—and technological change, 184 ff.; cultural, 301-29; medical, 330-1
Gertler, M. M., *et al.*, 144-5
Grossman, M. I., 340-1
Growth—of fingernails, 274-83; retardation of, during wartime, 258-73

Hansen, G. A., 231-2
*Hillman, R. W., Fingernail Growth: Rates and Variations, 274-83
Human resources of America, commission on, 147-9
*Hulse, F. S., Technological Advance and Major Racial Stocks, 184-92
Hunt, E. E., Jr., 233-4, 239-41

Impaired persons, life expectancy of, 145-7
Indians, American—blood groups among, 257; health of, 249-50

Japan—growth of school-age children in wartime, 258-63, 268; thoracic form in people of, 65-74

Keys, A., 144-5
Klepetar, E. H., 145-7
Klopsteg, P. E., and P. D. Wilson, 149-50
Kraus, B. S., 249-50

Larsson, T., and T. Sjögren, 150-2
Lasker, G. W., 241-3
*Lasker, M. W., Mortality of Persons with Xyloketosuria, 294-300
Learning, theory of, 50-1
*Lessa, W. A., Depopulation on Ulithi, 161-83
Liberty, biological basis of, 243-6
Life expectancy—in Chicago, 202 ff.; of impaired persons, 145-7; of persons with xyloketosuria, 294-300; of retired officers, 125 ff.
Limbs, artificial substitutes for, 149-50
Lull, L. J., 249-50

Markov process, 90 ff., 121 ff.

*Markowitz, S. D., Retardation in Growth of Children in Europe and Asia during World War II, 258-73
Martin, W. E., 239-41
*Mayer, A. J., Life expectancy in Chicago, 1880-1950, 202-10
McFarland, R. A., 247-8, 248-9
McLean, F. C., and M. R. Urist, 233-4
*McMahan, C. A., J. K. Folger and T. A. Ford, Longevity of Retired Army and Air Force Officers, 125-37
Medical—care, a bio-social study of, 232-3; genetics, 330-1
Meehl, P. E., 331-4
*Meier, P., Estimation in Markov Process with Constant Transition Rates, 321-4
Melanesia—absence of sickle-cell gene in some people of, 284-93; blood groups of, 187
Metabolism—anomaly of, and mortality, 294-300; standard values, 143
Migration, genetic effects of, 171 ff., 190
*Montagu, M. F. Ashley, 236-7; The Natchez Innominate Bone, 193-201
Mourant, A. E., 236-7, 253

*Nanda, R. S., Distribution Pattern of Dental Caries, 211-30
Natchez innominate bone, the, 193 ff.
Neel, J. V., and W. J. Schull, 334-5
*Newman, R. W., Skin-fold Changes with Increasing Obesity, 53-64
Nutrition—standard values in, 143; and growth in wartime, 260, 264-72

Obesity—and gain in weight, 39 ff.; and skin-fold changes, 53 ff.; patterns of, 75 ff.
*Ottensooser, F., Blood Groups, Races and Prehistory, 253-7

Paleoanthropology, 241-3; *see also* Fossil bones
Pentosuria, mortality of persons with, 294-300
Phenylthiourea, sensitivity to taste of, 153 ff.
Physiological—approach versus correlative analysis, 22 ff.; Congress, Nineteenth International, 340-1
Physique, an atlas of human, 336-40
Planansky, K., 150-2
*Pons, J.—Sexual Diagnosis of Isolated Bones, 12-21; Taste Sensitivity to Phenylthiourea in Spaniards, 153-60
Prediction, clinical versus statistical, 331-4
Prehistory and blood groups, 253-7
Psychiatric study of a rural population, 150-2
Psychological change, a study of, 237-9

Racial differences—and blood groups, 253 ff.; and technological advance, 184 ff.; in occurrence of sickle-cell gene, 284 ff.; in skin folds, 55 ff.; in taste sensitivity, 158-9
*Reid, D. B. W., and R. M. Grainger, Variations in the Caries Susceptibility of Children's Teeth, 1-11
*Ryan, R. E., Venereal Disease Contacts of Men, 32-8

Sheldon, W. H., C. W. Dupertuis and E. McDermott, 336-40
Sheps, C. G., and E. H. Taylor, 232-3
Sickle-cell gene, absence in some Melanesian populations, 284-93
Skin-fold changes and obesity, 53 ff.
Somatotyping, guide to, 336-40
Spuhler, J. N., 330-1
Stature—growth in wartime, 258 ff.; of men according to month of birth, 138-42
*Swindler, D. R., Absence of Sickle Cell Gene in Melanesian Societies, 284-93

*Takahashi, Eiji, and Hiroshi Atsumi, Age Differences in Thoracic Form, 65-74
Tanner, J. M., 336-40
Taste sensitivity to phenylthiourea, 153 ff.
Taylor, C. L., 149-50
Taylor, H. L., 232-3
Teeth, caries—in children, 1 ff.; distribution of, 211 ff.
Thoracic index, 65 ff.
Tobacco—biological effects of, 341-2; effect on taste of, 157
Twins, a study of, 235-6

Venereal disease—contacts, 32 ff.; depopulation resulting from, 176-8
Verschuer, O. von, 235-6

Weight—at birth during wartime, 268 ff.; gain and fat accumulation, 39 ff.
Willey, M. M., 147-9
Williams, R. J., 243-6
Wolfle, D., 147-9
Wynder, E. L., 341-2

Xyloketosuria, mortality of persons with, 294-300

*Zahl, S., Markov Process Model for Follow-up Studies, 90-120
Zubek, J. P., and P. A. Solberg, 237-9

INFORMATION FOR CONTRIBUTORS

HUMAN BIOLOGY, a record of research, is a quarterly journal devoted to the subject its name implies. It publishes original articles dealing with human genetics, development, growth, reproduction and aging, functional anatomy, bio-anthropology, human ecology, population dynamics, etc.

The original manuscripts, typed double-spaced on one side of 8½ x 11 inch paper in complete and finished form precisely as they are to appear, should be sent—packed flat—to the editor, Dr. Gabriel Lasker, Wayne University College of Medicine, 1401 Rivard Street, Detroit 7, Michigan. It is desirable that a second copy be included in order to expedite prompt consideration, and that an additional carbon copy and copies of all illustrations should be retained by the author.

Where possible, illustrations should be made for reproduction as line engravings, hence drawn in India ink as boldly as possible. Lettering must remain legible after figures are reduced to fit on 4½ x 6¾ inch pages (letters 1½ to 2 mm high after reduction are adequate). Figures should be numbered consecutively with Arabic numerals. Tables should be numbered independently from 1 up, and both figures and tables should have captions. When the amount of tabular and illustrative material is judged to be excessive or unusually expensive, authors may be requested to pay the excess cost. Galley proofs will be sent to the author and must be returned promptly; when the author makes substantial departures from copy, the cost will be charged to him. Three copies of an abstract (not to exceed 225 words) should be submitted when the proof is returned. One will be sent to the editor of *Biological Abstracts*.

References in the text to the literature should be made by using the author's name followed by the year of publication: "Studies of Garn and Saalberg ('53) . . ." At the end of the article, under the heading "LITERATURE CITED," full references with titles and inclusive page references should appear (double spaced) in alphabetical order of the authors' names: "GARN, S. M. AND J. H. SAALBERG 1953 Sex and age differences in the composition of the adult leg. Human Biol., 25: 144-153."

Authors will receive 25 free reprints. Orders for additional reprints should be placed at the time proof is returned. It will not be possible to accept orders for reprints after the proof has been returned to the press.

Human Biology

a record of research

VOLUME 27 DECEMBER, 1955 NUMBER 4

CONTENTS

BLOOD GROUPS, RACES AND PREHISTORY. *F. Ottensooser* 253

RETARDATION IN GROWTH OF CHILDREN IN EUROPE AND ASIA DURING WORLD WAR II. *Stephen D. Markowitz* 258

FINGERNAIL GROWTH IN THE HUMAN SUBJECT: RATES AND VARIATIONS IN 300 INDIVIDUALS. *Robert W. Hillman* 274

THE ABSENCE OF THE SICKLE CELL GENE IN SEVERAL MELANESIAN SOCIETIES AND ITS ANTHROPOLOGIC SIGNIFICANCE. *Doris R. Swindler* 284

MORTALITY OF PERSONS WITH XYLOKETOSURIA: A FOLLOW-UP STUDY OF A RARE METABOLIC ANOMALY. *Margaret Lasker* 294

A THEORY OF CULTURAL GENETICS. *Burt W. Aginsky* 301

BOOK REVIEWS 330

BOOK NOTICES 343

INDEX 347

WAYNE UNIVERSITY PRESS, *Publishers*

DETROIT 1, MICHIGAN, U. S. A.